PAPERGPT

ASRB EXAM 2026

VETERINARY PATHOLOGY

NODIA

NET, ARS, SMS (T-6) & STO (T-6)

CHAPTERWISE 25 YEARS PYQ

- 3000+ MCQs with Full Explanation
- As per syllabus released by Agricultural Scientists Recruitment Borad

PAPERGPT

Scan QR for Demo PDF

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

ASRB 2026

AGRICULTURAL SCIENTISTS RECRUITMENT BOARD

NET, ARS, SMS (T-6) & STO (T-6)

VETERINARY PATHOLOGY

Chapter-Wise Previous 25 Years PYQ Bank

PAPERGPT

Downloaded from www.pdf.tube

ASRB VETERINARY PATHOLOGY
Edition: July 2026
Copyright © By PAPERGPT

Information contained in this book has been obtained by author, from sources believes to be reliable. However, neither PAPERGPT nor its author guarantee the accuracy or completeness of any information herein, and PAPERGPT nor its author shall be responsible for any error, omissions, or damages arising out of use of this information. This book is published with the understanding that PAPERGPT and its author are supplying information but are not attempting to render engineering or other professional services.

NOIDIA

MRP ₹660.00

Published by :
PAPERGPT
125, Sector 6, Vidyadhar Nagar, Jaipur 302039
Phone :+91 9001005245

CONTENTS

CHAP : 01	Introduction, History and Etiology	7
CHAP : 02	Haemodynamics Derangements, Degeneration and Necrosis	52
CHAP : 03	Inflammation and Healing	95
CHAP : 04	Immunopathology	146
CHAP : 05	Genetically Determined Diseases	201
CHAP : 06	Disturbances in Cell Growth and Oncology	246
CHAP : 07	Postmortem Diagnosis and Histopathological Techniques	290
CHAP : 08	Clinical Pathology	334
CHAP : 09	Systemic Pathology	377
CHAP : 10	Pathology of Infectious Diseases	423
CHAP : 11	Avian Pathology	468
CHAP : 12	Nutritional and Production Pathology	513
CHAP : 13	Pathology of Toxicosis	556
CHAP : 14	Pathology of Diseases of Laboratory and Wild Animals	602

****

Click Here to Buy book on Amazon

30. VETERINARY PATHOLOGY

Unit 1: Introduction, History and Etiology

Introduction, history and scope of pathology. Definitions. Etiology of the disease. Predisposing factors, intrinsic and extrinsic factors responsible for the disease. Physical agents, mechanical injuries. Heat, cold and decreased atmospheric pressure, light (photosensitization) UV light, microwaves, electricity, chemical agents-exogenous chemicals (toxin, poisons, drugs and food substances), endogenous chemicals (metabolites, cytolytic or inhibitory immune complexes, free radicles, oxidants)

Unit 2: Haemodynamics Derangements, Degeneration and Necrosis

Disturbances of circulation/ haemodynamic derangements hyperaemia, ischaemia, haemorrhage, sludged blood, thrombosis, embolism, infarction, oedema and shock. Disturbances of cell metabolism – protein, carbohydrate and lipid metabolism, pigment metabolism, pathological calcification/ossification. Apoptosis, necrosis, gangrene. Ultrastructural changes in cell organelles in haemodynamic derangements and cell metabolic disturbances.

NODIA

Unit 3: Inflammation and Healing

Inflammation – definitions associated with inflammatory phenomenon, etiology of inflammation, cardinal signs, pathogenesis of inflammation, chemical mediators released from injured tissues and inflammatory cells. Cellular response in inflammation, structure and functions of cells associated with inflammation. Role of humoral and cell mediated defenses. Various classifications of inflammation. Healing, cellular regeneration capability of different body cells. Role of cells (macrophages, fibroblasts, myofibroblasts, endothelial cells), extracellular matrix components and growth factors in healing.

Unit 4: Immunopathology

Immunopathology – antibody and cells, immune-competence of foetus and new-born. Immune mediated tissue injury, hypersensitivity reactions- anaphylaxis, Arthus reaction, cyototoxic antibody reaction, immune complex disease, delayed hypersensitivity to chemicals, 78mmune-deficiency diseases, defective immuno- competence, autoimmune diseases.

Unit 5: Genetically Determined Diseases

Genetic abnormalities, aberrations of chromosomes, mosaicism, chimerism, anomalies in sex chromosomes and it autosomal chromosomes. Pathological states determined by one or more genes, lethal genes.

Unit 6: Disturbances in Cell Growth and Oncology

Disturbance in cell growth – aplasia, hypoplasia, hyperplasia, atrophy, metaplasia, dysplasia. Neoplasm: Etiology, carcinogens and oncogenesis, nomenclature and classification, characteristics of benign and malignant tumours, molecular mechanisms, pathways of spread of tumors and tumor immunology. Effects of tumour, grading, staging and laboratory diagnosis of tumours pathological features of various neoplasms.

Unit 7: Postmortem Diagnosis and Histopathological Techniques

Postmortem examination as a diagnostic tool. Postmortem techniques for different species including poultry, postmortem changes, lesions in various organs in different diseases, identification and interpretation of lesions, preparation of necropsy reports. Regulations dealing with diseases of animals in India regarding epidemiology, quarantine certificate, issue of soundness certificate. Handling of necropsy in vetero-legal cases, collection, preservation and dispatch of materials for diagnosis. Fixation and processing of tissues for histopathology and histochemistry. Different staining techniques. Histochemistry and histoenzymology as diagnostic tools. Principles of electron microscopy, processing of tissue for scanning and transmission electron microscopy.

Unit 8: Clinical Pathology

Clinical laboratory examination of various biomaterials from different livestock species, complete blood profile, changes in plasma/serum including biochemical profile for organ function tests, cytological examination and examination of urine, faeces, skin scrapings, cerebrospinal fluid and biopsy specimens and their interpretation.

Unit 9: Systemic Pathology

Advance pathology of cardiovascular, haemopoietic, respiratory, digestive, urinary, genital, nervous and musculoskeletal systems, endocrine glands, organ of special senses i.e, eye, ear, skin, appendages.

Unit 10: Pathology of Infectious Diseases

Etiology, pathology and pathogenesis of bacterial, viral, fungal, parasitic, mycoplasmal, rickettsial, chlamydial, and diseases caused by prions.

Unit 11: Avian Pathology

Omphalitis and yolk sac infection, Newcastle disease, infectious bronchitis, infectious laryngio-tracheitis, viral arthritis, infectious bursal disease, egg drop syndrome, inclusion body hepatitis and hydropericardium syndrome, infectious stunting syndrome, swollen head syndrome, Marek's disease, avian leucosis / sarcoma complex, salmonellosis, pasteurellosis, mycoplasmosis, chalamydiosis, colibacillosis, spirochaetosis, aspergillosis, thrush, mycotoxicosis, parasitic diseases - nematodes, cestodes and protozoa, nephrosis / nephritis syndrome, multi-etiology syndromes. Eio-pathology, clinical symptoms, and diagnosis of nutritional deficiencies - vitamin and mineral deficiencies; metabolic diseases- ascites, gout, fatty liver and kidney syndrome, fatty liver haemorrhagic syndrome, cage layer fatigue; miscellaneous conditions of poultry-heat stress, blue comb, breast blister, bumble foot, cannibalism, false layer, internal layer, pendulous crop, round heart disease. Emerging and re-emerging diseases of poultry.

Unit 12: Nutritional and Production Pathology

Pathology of nutritional deficiency disease - protein, carbohydrate, mineral and vitamins. Concept of production diseases - pathology of milk fever, ketosis, magnesium tetany, rumen indigestion, nutritional haemoglobinuria.

Unit 13: Pathology of Toxicosis

Pathology and pathogenesis of toxicosis due to heavy meals, mycotoxins, insecticides, pesticides, toxic plants, chemicals and drugs.

Unit 14: Pathology of Diseases of Laboratory and Wild Animals

Etiopathology of common diseases of laboratory, wild and zoo animals.

Downloaded from www.pdf.tube

NODIA

CHAPTER 01
Introduction, History and Etiology

2006
1. Which branch of veterinary pathology focuses primarily on the fundamental and basic mechanisms of disease at the cellular and tissue level?
(A) General pathology
(B) Systemic pathology
(C) Clinical pathology
(D) Surgical pathology

2021
2. The microscopic and macroscopic study of diseases concerning specific organ systems is formally known as:
(A) Molecular pathology
(B) Anatomic pathology
(C) Systemic pathology
(D) General pathology

2002
3. Which of the following statements correctly identifies the scope of clinical pathology?
(A) It analyzes blood, bodily fluids, and cytology to diagnose disease.
(B) It performs macroscopic and microscopic evaluations of surgical biopsies.
(C) It relies on post-mortem examinations of entire animal carcasses.
(D) It identifies genetic mutations and nucleic acid alterations in tissues.

2001
4. Identify the incorrect statement regarding the sub-divisions of veterinary pathology.
(A) Anatomic pathology includes both gross and microscopic examinations.
(B) Surgical pathology involves the examination of tissues removed from living animals.
(C) General pathology establishes the foundational principles of cell injury and inflammation.
(D) Molecular pathology is limited to the visual inspection of external lesions.

2001
5. Match the branches of pathology (List I) with their primary diagnostic medium (List II).
List I: (Branches of pathology)
P. Surgical pathology
Q. Clinical pathology
R. Molecular pathology
S. Anatomic pathology
List II: (Primary diagnostic medium)
1. Excised tissue from living patients
2. Blood, urine, and cavitary effusions
3. DNA, RNA, and targeted proteins
4. Whole body necropsy and organ evaluation
(A) P-1, Q-2, R-3, S-4
(B) P-2, Q-1, R-4, S-3
(C) P-3, Q-4, R-1, S-2
(D) P-4, Q-3, R-2, S-1

2008
6. Who is universally recognized as the "Father of Cellular Pathology" for determining that all diseases originate at the cellular level?
(A) John Hunter
(B) Giovanni Morgagni
(C) Rudolf Virchow
(D) Robert Koch

2001
7. The foundational concept that organs are the primary site of disease, establishing the basis for pathological anatomy, was introduced by which historical figure?
(A) Robert Koch
(B) Rudolf Virchow
(C) John Hunter
(D) Giovanni Morgagni

2024
8. The application of experimental pathology to study tissue repair and inflammation was heavily pioneered by:
(A) John Hunter
(B) Giovanni Morgagni
(C) Robert Koch
(D) Rudolf Virchow

2018
9. Which milestone in pathology is attributed strictly to Robert Koch?
(A) The development of high-resolution electron microscopes

Click Here to Buy book on Flipkart

(B) The formulation of postulates linking pathogens to diseases
(C) The discovery of the double-helical molecular structure of DNA
(D) The first systematic documentation of veterinary autopsies

2003

10. The concept of "omnis cellula e cellula" (every cell stems from another cell) was championed by:
(A) Giovanni Morgagni
(B) Rudolf Virchow
(C) John Hunter
(D) Robert Koch

2024

11. Given below are two statements, one labeled as Assertion (A) and the other as Reason (R).
Assertion (A): Giovanni Morgagni's major contribution to pathology was the creation of the germ theory of disease.
Reason (R): Morgagni focused on correlating clinical symptoms observed in life with anatomical changes found at autopsy.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is false but R is true.
(D) Both A and R are false.

2008

12. Which historical development directly enabled the rise of modern diagnostic histopathology?
(A) The development and widespread adoption of polymerase chain reactions
(B) The complete sequencing and mapping of the mammalian genome
(C) Refinements in light microscopy and tissue staining techniques
(D) The transition from gross macroscopic autopsies to advanced imaging

2001

13. A structural or functional alteration in a tissue resulting from disease is fundamentally defined as a:
(A) Symptom
(B) Prognosis
(C) Syndrome
(D) Lesion

2013

14. What term describes the step-by-step sequence of events detailing how a disease develops from inception to cellular manifestation?
(A) Prognosis
(B) Morbidity
(C) Pathogenesis
(D) Incubation

2025

15. A characteristic lesion that, when found, specifically and uniquely indicates a particular disease is known as:
(A) Idiopathic
(B) Pathognomonic
(C) Iatrogenic
(D) Congenital

2010

16. What is the primary distinction between an autopsy/necropsy and a biopsy?
(A) Necropsy involves advanced electron microscopy, whereas biopsy relies solely on macroscopic inspection.
(B) Necropsy is limited to checking biochemical fluids, while biopsy only evaluates solid parenchymal organs.
(C) Necropsy represents a specialized in vivo procedure, whereas biopsy is strictly performed post-mortem.
(D) Necropsy is a post-mortem exam, while biopsy evaluates tissue from a living patient.

2014

17. Identify the correct combination of statements regarding pathological artifacts.
I. Artifacts are artificially produced tissue changes that occur after death or during tissue processing.
II. Artifacts frequently serve as pathognomonic indicators of viral infections.
III. Recognizing artifacts is crucial to avoid misinterpreting them as true antemortem lesions.
Which of the above statements are correct?
(A) I and III
(B) II only
(C) II and III
(D) I and II

2001

18. Which of the following is considered an objective indication of disease perceptible to an examining veterinarian?
(A) Incubation
(B) Symptom
(C) Sign
(D) Prognosis

Downloaded from www.pdf.tube

2016
19. A subjective feeling or manifestation of disease recognized only by the patient (often difficult to definitively ascertain in veterinary medicine) is a:
(A) Symptom
(B) Morbidity
(C) Syndrome
(D) Sign

2001
20. A collection of concurrent signs and symptoms that together characterize a specific disease condition is termed a:
(A) Lesion
(B) Diagnosis
(C) Pathogenesis
(D) Syndrome

2010
21. Which term represents the scientific identification of a disease based on signs, lesions, and laboratory findings?
(A) Prognosis
(B) Diagnosis
(C) Artifact
(D) Morbidity

2006
22. The prediction of the probable course and outcome of a disease is known as the:
(A) Diagnosis
(B) Pathogenesis
(C) Prognosis
(D) Syndrome

2001
23. Which of the following represents the correct sequence of phases in the typical manifestation of a clinical infectious disease?
(A) Clinical signs, cell injury, exposure, structural alterations
(B) Exposure, cell injury, structural alterations, clinical signs
(C) Structural alterations, exposure, cell injury, clinical signs
(D) Exposure, incubation period, clinical signs, structural alterations

2010
24. The proportion of animals in a population that contract a particular disease over a specified period is termed:
(A) Morbidity
(B) Mortality
(C) Idiopathy
(D) Pathogenesis

2004
25. The ratio of deaths from a specific disease compared to the total number of individuals in the affected population is the definition of:
(A) Morbidity
(B) Mortality
(C) Prognosis
(D) Syndrome

NODIA

2023
26. The time elapsed between initial exposure to a pathogenic agent and the appearance of the first clinical signs is defined as the:
(A) Morbidity period
(B) Incubation period
(C) Prognostic window
(D) Pathogenic phase

2008
27. When classifying disease etiology, bacterial and viral pathogens fall into the category of:
(A) Animate causes
(B) Inanimate causes
(C) Genetic causes
(D) Idiopathic causes

2003
28. A nutritional deficiency is fundamentally classified as which type of etiological agent?
(A) Animate cause
(B) Idiopathic cause
(C) Inanimate cause
(D) Congenital cause

2024
29. Identify the incorrect combination of statements regarding disease etiology.
I. Acquired diseases are those exclusively caused by defective genes inherited from parents.
II. Congenital diseases are always strictly inherited and never caused by intrauterine environmental factors.
III. Idiopathic refers to a disease whose exact cause remains unknown.
IV. Animate causes include physical agents like extreme cold and radiation.
Which of the above statements are incorrect?
(A) I, II and IV
(B) II and III
(C) I, III and IV
(D) I and IV

2008

30. Diseases that develop at any point during an animal's life after birth, due to external agents, are classified as:
(A) Congenital
(B) Genetic
(C) Acquired
(D) Idiopathic

2012

31. An anatomical or physiological defect that is present precisely at birth is referred to as:
(A) Acquired
(B) Congenital
(C) Idiopathic
(D) Iatrogenic

2001

32. Which of the following represents a disease primarily caused by an intrinsic predisposing factor?
(A) Copper toxicity from contaminated pastures
(B) Frostbite from extreme winter exposure
(C) Mycotoxicosis from moldy feed
(D) Hip dysplasia driven by breed genetics

2014

33. An unusual, unpredictable individual hypersusceptibility or adverse reaction to a drug or chemical agent, unrelated to species or breed norms, is termed:
(A) Idiosyncrasy
(B) Congenital defect
(C) Sex linkage
(D) Nutritional imbalance

2004

34. The observation that specific coat color is strongly associated with the incidence of certain tumors (e.g., melanomas in gray horses) highlights which predisposing factor?
(A) Extrinsic environmental stress
(B) Iatrogenic chemical toxicity
(C) Intrinsic genetic factors
(D) Idiopathic external insults

2015

35. Match the following intrinsic predisposing factors (List I) with an appropriate example (List II).
List I: (Intrinsic predisposing factors)
P. Species
Q. Breed
R. Age
S. Sex
List II: (Examples)
1. Greater incidence of prostatic hyperplasia in males
2. Higher occurrence of osteoarthritis in geriatric animals
3. Canine distemper virus affecting dogs but not cattle
4. Brachycephalic airway syndrome in Pugs
(A) P-4, Q-3, R-1, S-2
(B) P-2, Q-1, R-4, S-3
(C) P-3, Q-4, R-2, S-1
(D) P-1, Q-2, R-3, S-4

2008

36. Which of the following best describes an extrinsic predisposing factor to disease?
(A) Immune suppression related specifically to aging
(B) A highly specific sex-linked genetic abnormality
(C) Anatomical breed conformation traits and habits
(D) Climatic stress from extreme weather

2009

37. Nutritional imbalances and deficiencies act as major predisposing factors. Which condition results primarily from a severe deficiency of dietary energy and protein over time?
(A) Obesity
(B) Starvation
(C) Iatrogenic toxicity
(D) Photosensitization

2001

38. Poor ventilation and overcrowding in livestock housing are classic examples of:
(A) Intrinsic genetic defects
(B) Animate pathogen mutations
(C) Managemental faults
(D) Idiopathic predisposing factors

2014

39. How do chronic managemental faults, such as overcrowding, primarily predispose animals to infectious disease outbreaks?
(A) By altering the baseline genetic sequence of the host
(B) By mutating the coat color expression across the herd
(C) By triggering an acute idiosyncratic systemic drug reaction
(D) By increasing stress and pathogen exposure

2006

40. A scrape removing the superficial epithelial layer of the skin due to friction is categorized pathologically as an:

Click Here to Buy book on Amazon

(A) Abrasion
(B) Incision
(C) Laceration
(D) Contusion

2017

41. Identify the correct combination of statements concerning mechanical injuries.
I. A contusion results from blunt force trauma that ruptures blood vessels without breaking the skin.
II. An incision is a clean cut made by a sharp-edged object.
III. Abrasions involve deep, irregular tearing of subcutaneous tissues.
IV. Punctures are narrow, deep wounds caused by sharp, pointed instruments.
V. A fracture involves the mechanical discontinuity of a bone.
Which of the above statements are correct?
(A) I, II, IV and V
(B) II, III and V
(C) I, III and IV
(D) I, II, III and IV

2017

42. An irregular, torn wound caused by a tearing force that disrupts skin and underlying tissue is termed a:
(A) Contusion
(B) Laceration
(C) Puncture
(D) Abrasion

2014

43. A clean, smooth-edged wound intentionally created by a scalpel during surgery is best classified as an:
(A) Incision
(B) Abrasion
(C) Contusion
(D) Laceration

2021

44. What type of mechanical injury is characterized by a focal, deep penetration of tissue, commonly predisposing the animal to anaerobic bacterial infections like tetanus?
(A) Contusion
(B) Abrasion
(C) Puncture
(D) Incision

NODIA

2023

45. A break in the continuity of bone or cartilage caused by excessive mechanical stress is formally known as a:
(A) Contusion
(B) Laceration
(C) Puncture
(D) Fracture

2005

46. What is the defining characteristic of a third-degree thermal burn?
(A) Mild active erythema restricted completely to the epidermis
(B) Extensive subcutaneous edema without any epidermal damage
(C) Full-thickness destruction of skin and local nerves
(D) Isolated intraepidermal vesicle formation with minimal scarring

2010

47. Arrange the following stages of systemic heat stroke pathogenesis in the correct order:
I. Extensive systemic protein denaturation
II. Prolonged exposure to high ambient temperature and humidity
III. Multiorgan failure and death
IV. Failure of thermoregulatory mechanisms and severe hyperthermia
Which of the following represents the correct sequence?
(A) II, I, IV, III
(B) I, IV, II, III
(C) II, IV, I, III
(D) IV, I, II, III

2008

48. Heat exhaustion is primarily mediated by which immediate physiological failure?
(A) Complete macroscopic destruction of deep skeletal bone tissues
(B) Extensive necrosis of subcutaneous adipose tissues and fat cells
(C) Widespread irreversible denaturation of DNA within circulating lymphocytes
(D) Profound peripheral vasodilation leading to circulatory shock

2010

49. The fundamental mechanism of local cellular injury during exposure to extreme localized heat is:
(A) Protein denaturation and coagulation
(B) Rapid crystallization of intracellular water
(C) Intracellular accumulation of lipofuscin

pigment
(D) Systemic inhibition of phylloerythrin excretion

2009
50. First-degree burns are clinically characterized primarily by:
(A) Charring of underlying muscle
(B) Coagulation of full-thickness skin
(C) Erythema and mild pain
(D) Complete loss of sensory nerves

2005
51. The primary local tissue damage resulting from severe extreme cold exposure (frostbite) is caused by:
(A) Ice crystal formation and thrombosis-induced ischemia
(B) Severe electroporation and physical tearing of host cell membranes
(C) Immediate pyrimidine dimer formation within epidermal nuclei
(D) Rapid thermal denaturation of critical structural cytoproteins

2021
52. Systemic exposure to abnormally low environmental temperatures resulting in a life-threatening drop in core body temperature is defined as:
(A) Heat stroke
(B) Photosensitization
(C) Altitude sickness
(D) Hypothermia

2010
53. Given below are two statements, one labeled as Assertion (A) and the other as Reason (R).
Assertion (A): Prolonged severe frostbite frequently results in ischemic necrosis and gangrene of extremities.
Reason (R): Extreme cold induces severe vasoconstriction followed by endothelial damage and intravascular thrombosis.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2013
54. In high-altitude environments, the primary pathological stressor on an animal's body is:
(A) Increased atmospheric pressure leading to gas embolism
(B) Excessive environmental oxygen causing oxidative stress
(C) Low atmospheric pressure causing systemic hypoxia
(D) Severe blockage of solar ultraviolet radiation

2016
55. "Brisket disease" in cattle grazing at high altitudes is primarily an expression of:
(A) Left-sided heart failure due to aortic stenosis
(B) Liver failure from primary photosensitization
(C) Acute renal failure due to extreme drops in atmospheric pressure
(D) Right-sided heart failure from pulmonary hypertension

2011
56. What is the foundational pathological change in the pulmonary vasculature of animals suffering from chronic altitude sickness?
(A) Medial hypertrophy of pulmonary arterioles
(B) Diffuse pulmonary artery thinning and pathological dilation
(C) Endothelial disruption accompanied by alveolar hemorrhage
(D) Accumulation of toxic heavy metals in vascular endothelium

2025
57. High-altitude disease in cattle frequently manifests with severe, dependent subcutaneous edema located prominently in the:
(A) Dorsal thoracic area
(B) Brisket region
(C) Cranial cavity
(D) Interdigital space

2001
58. Photosensitization requires three factors: a photodynamic substance in the skin, exposure to specific wavelengths of light, and:
(A) High environmental humidity and high ambient temperature
(B) Lightly pigmented or unpigmented skin
(C) A concurrent secondary bacterial skin infection
(D) Acute systemic heavy metal toxicity

2011
59. Identify the incorrect combination of statements regarding photosensitization.
I. Primary photosensitization occurs when a preformed photodynamic agent is ingested and absorbed directly into circulation.
II. Secondary (hepatogenous) photosensitization is caused by liver damage preventing the

Downloaded from www.pdf.tube

excretion of phylloerythrin.
III. Congenital porphyria is an infectious viral disease that destroys melanin.
Which of the above statements are incorrect?
(A) I and II
(B) II only
(C) I and III
(D) III only

2015
60. Hepatogenous photosensitization in ruminants is most commonly linked to the accumulation of which circulating compound?
(A) Porphobilinogen
(B) Superoxide dismutase
(C) Phylloerythrin
(D) Melanin

2008
61. Phylloerythrin is a photodynamic agent that is normally formed in the gastrointestinal tract by microbial breakdown of:
(A) Hemoglobin
(B) Myoglobin
(C) Bilirubin
(D) Chlorophyll

2002
62. Which type of photosensitization is strictly due to an inherited metabolic defect in the synthesis of heme, resulting in the accumulation of porphyrins?
(A) Primary photosensitization
(B) Hepatogenous photosensitization
(C) Congenital porphyria
(D) Contact photosensitization

2004
63. Non-ionizing radiation, such as ultraviolet (UV) light, primarily damages cells by causing the formation of:
(A) Double-strand DNA breaks via massive water radiolysis
(B) Pyrimidine (thymine) dimers in DNA
(C) Massive heat coagulation of tissue
(D) Lichtenberg figures in the dermis

2010
64. Chronic exposure to UV radiation is a well-known extrinsic predisposing factor for which specific neoplasm in lightly pigmented animals?
(A) Osteosarcoma of the appendicular skeleton
(B) Primary hepatic adenocarcinoma
(C) Multicentric malignant lymphoma
(D) Squamous cell carcinoma

2009
65. Match the following physical agents (List I) with their primary pathological consequence (List II).
List I: (Physical agents)
P. Cold (Frostbite)
Q. Altitude (Hypoxia)
R. UV Light
S. Extreme Heat
List II: (Primary pathological consequences)
1. Pulmonary hypertension
2. DNA pyrimidine dimers
3. Ischemic necrosis via thrombosis
4. Protein denaturation
(A) P-3, Q-1, R-2, S-4
(B) P-4, Q-2, R-1, S-3
(C) P-3, Q-2, R-1, S-4
(D) P-1, Q-4, R-3, S-2

NODIA

2017
66. Actinic keratosis is a precancerous epidermal lesion primarily induced by prolonged exposure to:
(A) Microwaves
(B) Gamma rays
(C) UV radiation
(D) Extreme cold

2015
67. The fundamental tissue damage inflicted by ionizing radiation (like X-rays and gamma rays) is largely mediated by:
(A) Radiolysis of cellular water, generating free radicals
(B) Direct formation of pyrimidine dimers within host cell nuclei
(C) Denaturation of structural skin proteins via localized thermal heat
(D) Severe localized hypothermia in targeted parenchymal tissues

2014
68. Which cell population is most acutely vulnerable to the destructive effects of ionizing radiation?
(A) Fully mature and differentiated neurons of the cerebral cortex
(B) Highly differentiated cardiac muscle myocytes of the myocardium
(C) Well-differentiated mature skeletal muscle fibers
(D) Dividing cells in bone marrow and intestinal crypts

2009
69. High-dose whole-body exposure to ionizing radiation leads to Acute Radiation Syndrome, which most commonly manifests first as:

(A) Sudden and uncontrolled massive osteoblast-driven bone overgrowth
(B) Bone marrow suppression and gastrointestinal sloughing
(C) Immediate and widespread necrotic damage to skeletal muscle tissues
(D) Severe congenital malformations and systemic structural anomalies

2025

70. Microwave radiation primarily causes tissue injury through which mechanism?
(A) Direct genomic mutation via double-stranded DNA backbone breaks
(B) Systemic cutaneous accumulation of photodynamic phylloerythrin
(C) Internal heating from agitation of water molecules
(D) Massive intravascular ice crystallization and physical cell rupture

2001

71. Identify the correct combination of statements concerning electrical and lightning injuries.
I. Alternating current generally causes more severe injury than direct current of the same voltage because it induces tetanic muscle spasms.
II. The primary cause of death in severe electrical shock is usually ventricular fibrillation or respiratory paralysis.
III. Lightning strictly produces freezing injuries due to extreme atmospheric changes.
IV. Burns typically occur at the points where the electrical current enters and exits the body.
Which of the above statements are correct?
(A) I, II and III
(B) II, III and IV
(C) I, II and IV
(D) I and IV

2019

72. Branching, fern-like erythematous skin marks sometimes observed on the skin of animals struck by lightning are called:
(A) Lichtenberg figures
(B) Pyrimidine dimer lesions
(C) Frostbite line markings
(D) Actinic keratosis tracks

2008

73. High-voltage electrical injury can induce direct cellular damage independent of thermal burns by creating pores in cell membranes, a process called:
(A) Coagulation
(B) Electroporation
(C) Radiolysis
(D) Porphyria

2006

74. Which of the following heavy metals classically causes toxicity characterized by encephalopathy and the formation of acid-fast intranuclear inclusion bodies in renal tubular epithelium?
(A) Copper
(B) Lead
(C) Arsenic
(D) Mercury

2007

75. Chronic toxicity of which element in sheep is famously associated with sudden massive intravascular hemolysis, resulting in a dark "gunmetal" colored kidney?
(A) Copper
(B) Mercury
(C) Arsenic
(D) Lead

2006

76. Inorganic arsenic toxicity primarily targets which anatomical structure, resulting in profound fluid loss and hemorrhagic diarrhea?
(A) The myelin sheath of peripheral motor and sensory nerves
(B) The delicate rods and cones within the sensory retina
(C) Capillary endothelium, especially in the GI tract
(D) Pulmonary alveolar macrophages within distal airways

2021

77. Which sequence properly orders the typical manifestation of chronic copper toxicosis in sheep?
I. Hepatic accumulation of copper over months
II. Sudden stressful event acting as a trigger
III. Acute massive intravascular hemolysis and hemoglobinuria
IV. Massive release of stored copper into the bloodstream
Which of the following represents the correct sequence of these events?
(A) I, II, IV, III
(B) I, IV, II, III
(C) II, I, III, IV

Click Here to Buy book on Flipkart

(D) IV, I, II, III

2023

78. Mercury poisoning primarily causes significant pathological damage to the:
(A) Exocrine pancreas and pancreatic ducts
(B) Epidermal layers and hair follicle cells
(C) Central nervous system and renal tubules
(D) Articular cartilage of major synovial joints

2025

79. Mycotoxicosis is a form of exogenous organic toxicity caused by the ingestion of food or feed contaminated by toxins produced by:
(A) Bacteria
(B) Fungi
(C) Viruses
(D) Poisonous plants

2009

80. Aflatoxicosis, caused by ingestion of toxins from Aspergillus flavus, most prominently targets which organ?
(A) Heart
(B) Lungs
(C) Spleen
(D) Liver

2014

81. The ingestion of Bracken fern (Pteridium aquilinum) in cattle is notoriously associated with toxic destruction of the bone marrow and the development of:
(A) Urinary bladder neoplasms and hematuria
(B) Chronic bleeding ulcers of the gastric mucosa
(C) Progressive bilateral outer retinal degeneration
(D) Acute diffuse interstitial pulmonary emphysema

2007

82. Which statement correctly distinguishes neurotoxic from hemotoxic snake venoms?
(A) Neurotoxins cause widespread dermal tissue necrosis, while hemotoxins strictly target and paralyze the diaphragm.
(B) Neurotoxins solely affect red blood cell production, while hemotoxins attack cellular elements of the cerebral cortex.
(C) Neurotoxins block neuromuscular transmission, while hemotoxins cause coagulopathy and tissue damage.
(D) Neurotoxins induce mechanical bone fractures, whereas hemotoxins trigger acute toxic liver and pancreatic failure.

2014

83. Given below are two statements, one labeled as Assertion (A) and the other as Reason (R).
Assertion (A): Secondary (hepatogenous) photosensitization is caused by direct ingestion of photodynamic plants like St. John's wort.
Reason (R): Liver failure prevents the clearance of normal metabolic porphyrins, causing them to accumulate in the skin.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2009

84. Ergotism, a toxicosis caused by the ingestion of Claviceps purpurea mycotoxins on grain, primarily causes:
(A) Primary hepatocellular carcinoma with pulmonary metastasis
(B) Massive internal hemorrhage into the peritoneal cavity
(C) Acute progressive renal tubular failure and uremia
(D) Severe vasoconstriction causing gangrene of extremities

2012

85. Tissue damage resulting inadvertently from therapeutic drugs or medical treatments administered by a veterinarian is classified as:
(A) Congenital anomaly
(B) Iatrogenic toxicity
(C) Idiopathic disease
(D) Animate infection

2012

86. Which class of therapeutic antibiotics is notoriously associated with iatrogenic nephrotoxicity and ototoxicity if administered in excessive doses?
(A) Penicillins
(B) Macrolides
(C) Aminoglycosides
(D) Cephalosporins

NODIA

2002

87. Organophosphate agricultural chemicals cause severe toxicity primarily by:
(A) Completely destroying the mucosal epithelial lining of the stomach
(B) Directly inducing massive intravascular hemolysis of red blood cells
(C) Blockading voltage-gated slow calcium

channels in the myocardium
(D) Inhibiting acetylcholinesterase, causing cholinergic excess

2022

88. Anticoagulant rodenticides cause fatal internal hemorrhage by competitively antagonizing which essential vitamin?
(A) Vitamin K
(B) Vitamin C
(C) Vitamin D
(D) Vitamin E

2024

89. Identify the incorrect combination of statements regarding toxic food additives and adulterants.
I. Melamine toxicity causes severe acute renal failure via crystal formation in renal tubules.
II. Melamine is an essential dietary amino acid supplemented to boost protein.
III. Excessive dietary urea in ruminants can lead to fatal ammonia toxicosis.
IV. Food adulteration is exclusively a problem of inanimate plant toxins naturally occurring in feed.
Which of the above statements are incorrect?
(A) I, II and III
(B) I and III
(C) II and IV
(D) II, III and IV

2024

90. Endogenous metabolic chemicals can cause severe tissue injury. Elevated blood urea nitrogen (BUN) and creatinine indicate uremia, which commonly results in:
(A) Osteosarcoma of the jaw
(B) Stomatitis and gastropathy
(C) Diffuse hepatic necrosis
(D) Bilateral corneal ulceration

2024

91. Ketoacidosis is a severe endogenous metabolic condition characterized by the pathological overproduction of ketone bodies, most commonly seen as a complication of:
(A) Diabetes mellitus or negative energy balance
(B) Acute systemic heavy metal toxicosis from lead or mercury
(C) Subacute or chronic radiation syndrome
(D) Hypervolemia caused by overhydration

2014

92. The yellow discoloration of mucous membranes, sclera, and tissues due to excessive accumulation of bilirubin in the blood is known as:
(A) Congenital porphyria
(B) Pathological melanosis
(C) Icterus (Jaundice)
(D) Systemic cyanosis

NODIA

2023

93. Excessive breakdown of erythrocytes, overwhelming the liver's capacity to conjugate bilirubin, leads directly to:
(A) Hepatic icterus
(B) Post-hepatic (obstructive) icterus
(C) Uremic icterus
(D) Pre-hepatic (hemolytic) icterus

2021

94. Post-hepatic icterus is primarily caused by:
(A) Massive intravascular hemolysis of circulating erythrocytes
(B) Direct toxic damage to hepatocytes from chemicals
(C) Bile duct obstruction preventing bilirubin excretion
(D) Severe chronic deficiency of essential dietary iron

2014

95. Match the form of icterus (List I) with its primary underlying mechanism (List II).
List I: (Forms of icterus)
P. Pre-hepatic icterus
Q. Hepatic icterus
R. Post-hepatic icterus
S. Neonatal icterus (Isoerythrolysis)
List II: (Primary underlying mechanisms)
1. Biliary tract obstruction
2. Massive erythrocyte destruction
3. Maternal antibody-mediated hemolysis
4. Hepatocellular damage/necrosis
(A) P-4, Q-3, R-1, S-2
(B) P-2, Q-4, R-1, S-3
(C) P-1, Q-2, R-3, S-4
(D) P-3, Q-1, R-2, S-4

2018

96. Type II hypersensitivity reactions mediate tissue injury fundamentally through:
(A) Deposition of circulating immune complexes in small vessel walls
(B) Immediate IgE-mediated tissue mast cell degranulation and histamine release
(C) T-cell mediated delayed-type hypersensitivity and cytotoxicity
(D) Antibodies binding directly to cell surface antigens

2004
97. Which of the following mechanisms is classically utilized in Type II hypersensitivity to destroy targeted host cells?
(A) Complement Membrane Attack Complex causing cell lysis
(B) Chronic granuloma formation driven by activated tissue macrophages
(C) Massive systemic histamine release from activated tissue mast cells
(D) The formation of pyrimidine dimers within local epidermal nuclei

2016
98. Antibody-dependent cellular cytotoxicity (ADCC) is a process in which:
(A) Cytotoxic T-cells destroy target cells without any prior antibody sensitization
(B) Polymorphonuclear neutrophils undergo necrosis upon ingesting large bacteria
(C) Leukocytes destroying target cells coated with specific antibodies
(D) Excess free radicals spontaneously lysing circulating mature red blood cells

2007
99. Which clinical disease is a classic example of a cytolytic Type II hypersensitivity reaction?
(A) Autoimmune hemolytic anemia
(B) Chronic rheumatoid arthritis
(C) Systemic tuberculin reaction
(D) Localized anaphylactic shock

2008
100. Neonatal isoerythrolysis in foals occurs when the newborn absorbs colostral antibodies that attack its own red blood cells. This is a manifestation of:
(A) Type I hypersensitivity
(B) Type III hypersensitivity
(C) Type IV hypersensitivity
(D) Type II hypersensitivity

2005
101. Arrange the sequence of events leading to cellular destruction via complement-mediated Type II hypersensitivity:
I. Formation of the Membrane Attack Complex (MAC)
II. Binding of IgG or IgM to a cell surface antigen
III. Osmotic lysis of the target cell
IV. Activation of the classical complement cascade
Which of the following represents the correct sequence of these events?
(A) II, IV, I, III
(B) I, II, IV, III
(C) IV, I, III, II
(D) II, I, IV, III

2017
102. What is the fundamental pathological mechanism of Type III hypersensitivity?
(A) Cytolytic destruction of host cells by direct binding of specific antibodies
(B) Chronic granulomatous inflammation driven primarily by $CD4^+$ TH1 cells
(C) Deposition of circulating soluble immune complexes in tissues
(D) Immediate systemic release of vasoactive histamine from tissue mast cells

2019
103. In Type III hypersensitivity, where do immune complexes most frequently deposit to cause severe tissue damage?
(A) Large muscular arteries, deep subcutaneous fat, and bone cortex
(B) Cartilage, tendons, and ligamentous insertions
(C) Epidermal layers and mucosal surfaces
(D) Small blood vessel walls, renal glomeruli, and synovial joints

2018
104. The classic localized form of Type III hypersensitivity, characterized by severe dermal edema, hemorrhage, and necrosis at the site of repeated antigen injection, is called:
(A) Arthus reaction
(B) Acute anaphylaxis
(C) Systemic serum sickness
(D) Actinic keratosis lesion

2024
105. Serum sickness is best described as a:
(A) Localized Type I immediate hypersensitivity
(B) Systemic Type III reaction
(C) Delayed-type Type IV hypersensitivity
(D) Cytolytic-type Type II hypersensitivity

2008
106. In immune complex-mediated (Type III) glomerulonephritis, the severe local tissue damage is ultimately executed by:
(A) Cytotoxic T-lymphocytes directly attacking and destroying podocytes
(B) Massive histamine and cytokine release from tissue-resident mast cells
(C) Lysosomal enzymes and reactive oxygen species from recruited neutrophils

Downloaded from www.pdf.tube

(D) Fungal mycotoxins binding directly to basement membrane components

2013

107. Consider the following statements concerning Type III hypersensitivity:

I. It is exclusively mediated by IgE antibodies.
II. Immune complex deposition often leads to vasculitis characterized by fibrinoid necrosis.
III. Activation of the complement cascade by immune complexes is a critical step in recruiting inflammatory cells.

Which of the above statements is/are correct?

(A) I and II
(B) I and III
(C) I only
(D) II and III

2019

108. Fibrinoid necrosis of blood vessels is a hallmark histological finding of which immune-mediated tissue injury?

(A) Type I hypersensitivity
(B) Type II hypersensitivity
(C) Type III hypersensitivity
(D) Type IV hypersensitivity

2015

109. What defines a free radical?

(A) A highly unstable compound completely devoid of all orbital electrons
(B) Any chemical species with an unpaired electron in its outer orbit
(C) A highly stable compound possessing a fully paired outer electron shell
(D) A negatively charged ionic compound completely bound to sodium atoms

2010

110. Reactive Oxygen Species (ROS) are primarily generated endogenously during which normal physiological process?

(A) Anaerobic glycolysis in the cytoplasm
(B) Mitochondrial oxidative phosphorylation
(C) Ribosomal protein translation of mRNA
(D) Mitotic spindle assembly and separation

2019

111. Which of the following is a free radical species produced by the univalent reduction of molecular oxygen?

(A) Superoxide anion ($O_2^{\cdot -}$)
(B) Hydrogen peroxide (H_2O_2)
(C) Water (H_2O)
(D) Ozone (O_3)

2007

112. Hydrogen peroxide (H_2O_2) is technically not a free radical itself, but it is highly pathogenic because it can be converted into the highly reactive hydroxyl radical ($OH^{\cdot}$) via the:

(A) Krebs cycle
(B) Cori cycle
(C) Fenton reaction
(D) Calvin cycle

2002

113. Given below are two statements, one labeled as Assertion (A) and the other as Reason (R).

Assertion (A): Hydrogen peroxide is considered a biologically harmless byproduct of metabolism.
Reason (R): It acts as a direct free radical that immediately destroys the cell membrane without enzymatic intervention.

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) Both A and R are false.

2017

114. The Fenton reaction primarily utilizes which transitional metal ion to catalyze the formation of hydroxyl radicals from hydrogen peroxide?

(A) Calcium (Ca^{2+})
(B) Zinc (Zn^{2+})
(C) Iron (Fe^{2+})
(D) Magnesium (Mg^{2+})

2021

115. Which of the following is NOT a primary mechanism by which free radicals induce cellular injury?

(A) Massive lipid peroxidation of cellular and organelle membranes
(B) Direct inhibition of ATP synthase via competitive enzyme binding
(C) Oxidative modification and covalent cross-linking of cellular proteins
(D) Continuous formation of single- and double-strand breaks in DNA

NODIA

2014

116. Lipid peroxidation destroys cell membranes because free radicals aggressively steal electrons from the double bonds of:

(A) Hydrophobic saturated fatty acids
(B) Branched cellular glycogen chains
(C) Structurally active glycoproteins
(D) Membrane polyunsaturated fatty acids

2008

117. What is a common, measurable end-product of lipid peroxidation frequently used as a biomarker for oxidative stress?
(A) Malondialdehyde (MDA)
(B) Pyruvic acid
(C) Uric acid
(D) Beta-hydroxybutyrate

2013

118. How do reactive oxygen species fundamentally damage cellular proteins?
(A) By systematically replacing normal peptide bonds with glycosidic linkages
(B) By directly converting hydrophobic amino acid residues into saturated fats
(C) By promoting sulfhydryl-mediated cross-linking and fragmentation
(D) By rapidly dissolving complex structural proteins into pure water molecules

2011

119. Consider the following statements regarding oxidative DNA damage:
I. Free radicals frequently cause single- and double-strand breaks in nuclear DNA.
II. Oxidative DNA damage is irrelevant to cellular aging or neoplastic transformation.
III. The hydroxyl radical is the primary mediator of severe DNA oxidative damage.
IV. Oxidative stress strictly enhances genomic stability by strengthening nucleotide bonds.
Which of the above statements is/are INCORRECT?
(A) II and IV
(B) I and III
(C) II and III
(D) I and IV

2015

120. Which of the following best describes the physiological consequence of extensive protein cross-linking induced by free radicals?
(A) Prominent enhancement of cellular enzymatic efficiency and output
(B) Markedly accelerated mitotic cell division and tissue regeneration
(C) Significant increase in overall lipid bilayer membrane fluidity
(D) Enzymatic inactivation and degradation of vital structural proteins

2009

121. The enzymatic antioxidant defense system utilizes specific scavengers to neutralize ROS. Which enzyme specifically converts superoxide anions ($O_2^{\cdot -}$) into hydrogen peroxide and oxygen?
(A) Catalase
(B) Superoxide dismutase (SOD)
(C) Glutathione peroxidase
(D) Myeloperoxidase

2023

122. Which endogenous cellular enzyme, predominantly located in peroxisomes, decomposes hydrogen peroxide into water and oxygen?
(A) Cytochrome C oxidase
(B) Superoxide dismutase
(C) Catalase
(D) Lactate dehydrogenase

2010

123. Glutathione peroxidase is a vital intracellular antioxidant enzyme that detoxifies lipid peroxides and hydrogen peroxide. It crucially requires which trace mineral as a cofactor?
(A) Copper
(B) Iron
(C) Calcium
(D) Selenium

2004

124. Which of the following is a major lipid-soluble non-enzymatic antioxidant that protects cellular membranes against lipid peroxidation by acting as a free radical chain breaker?
(A) Vitamin E (alpha-tocopherol)
(B) Vitamin B12 (cobalamin)
(C) Vitamin C (ascorbic acid)
(D) Folic acid (folate)

2004

125. Match the antioxidant agent in List I with its primary cellular location or role in List II.
List I: Antioxidant agent
P. Superoxide dismutase
Q. Catalase
R. Vitamin E
S. Vitamin C
List II: Primary cellular location or role
1. Cell membranes (lipid-soluble scavenger)
2. Cytosol and mitochondria (converts superoxide)
3. Aqueous phase (water-soluble scavenger)
4. Peroxisomes (decomposes hydrogen peroxide)
(A) P-4, Q-2, R-3, S-1
(B) P-2, Q-4, R-1, S-3
(C) P-1, Q-3, R-2, S-4

Click Here to Buy book on Amazon

(D) P-3, Q-1, R-4, S-2

2016

126. Vitamin C (ascorbic acid) functions as a crucial antioxidant primarily by:
(A) Directly inserting into hydrophobic lipid bilayers to block peroxidation
(B) Scavenging aqueous free radicals and regenerating oxidized Vitamin E
(C) Enzymatically converting superoxide anions into cellular hydrogen peroxide
(D) Serving as an essential structural and functional component of genomic DNA

2010

127. A profound deficiency of Vitamin E and Selenium in livestock typically leads to severe oxidative tissue damage, famously presenting as:
(A) Nutritional muscular dystrophy (White muscle disease)
(B) Primary photodynamic dermatitis (Photosensitization)
(C) Systemic amyloidosis involving internal visceral organs
(D) Acute toxicosis from lead or other heavy metal poisoning

2004

128. Nitric oxide (NO) can act both as a signaling molecule and a destructive free radical. When NO reacts directly with the superoxide anion, it forms which highly reactive intermediate?
(A) Hydrogen peroxide
(B) Hydroxyl radical
(C) Peroxynitrite ($ONOO^-$)
(D) Singlet oxygen

2018

129. The term "oxidative stress" refers fundamentally to a cellular state where:
(A) The complete consumption of all available oxygen within the cell
(B) Excessive activity of antioxidant enzymes in the absence of free radicals
(C) Unusually rapid diffusion of oxygen across the blood-air barrier
(D) Reactive oxygen species generation overwhelming cellular antioxidant defenses

2001

130. Why is the central nervous system particularly susceptible to oxidative stress and free radical injury?
(A) It entirely lacks functional mitochondria and other metabolic organelles.
(B) It functions effectively under anaerobic conditions without any oxygen.
(C) It has high oxygen use and membranes rich in polyunsaturated lipids.
(D) It actively synthesizes and secretes massive amounts of heavy metals.

2002

131. Consider the following statements concerning cellular antioxidant defenses:
I. Catalase is highly concentrated in lysosomes.
II. Glutathione peroxidase requires selenium to function efficiently.
III. Vitamin E protects lipid membranes from peroxidation.
IV. Superoxide dismutase converts superoxide into hydrogen peroxide.
V. Vitamin C strictly promotes the generation of hydroxyl radicals.
Which of the above statements is/are correct?
(A) I, II and V
(B) II, III and IV
(C) I, III and IV
(D) I, II, III and V

2014

132. The process by which tissues are preserved immediately after death or removal to prevent autolysis and preserve morphology is termed:
(A) Biopsy
(B) Maceration
(C) Putrefaction
(D) Fixation

2004

133. A definitive sign of absolute, irreversible cell death at the microscopic level is:
(A) Nuclear dissolution (Karyolysis)
(B) Swelling of the endoplasmic reticulum
(C) Clumping of nuclear chromatin
(D) Mild cellular edema

NODIA

2008

134. Which foundational concept establishes that changes at the microscopic cellular level are directly responsible for the gross clinical signs of disease?
(A) Humoral pathology
(B) Idiopathic pathophysiology
(C) Cellular pathology
(D) Congenital morphology

2013

135. The branch of pathology focused on diagnosing disease at the gene, RNA, and protein level using techniques like PCR and immunohistochemistry is:

(A) Molecular pathology
(B) Gross pathology
(C) Surgical pathology
(D) Forensic pathology

2012

136. What distinguishes a strictly idiopathic disease from a well-characterized acquired disease?
(A) The acquired disease has an unknown origin, whereas the idiopathic disease is caused exclusively by viruses.
(B) The idiopathic disease is restricted to development in utero, while the acquired disease occurs postnatally.
(C) The idiopathic disease is always immediately fatal, whereas the acquired disease is mild and self-limiting.
(D) The idiopathic disease has an unknown etiology, whereas the acquired disease has a defined external cause.

2002

137. Given below are two statements, one labeled as Assertion (A) and the other as Reason (R).
Assertion (A): Starvation serves as a profound extrinsic predisposing factor for infectious diseases.
Reason (R): Severe nutritional deficiency severely compromises the host's immune system, diminishing resistance to animate pathogens.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2023

138. Prolonged physical compression or crushing of skeletal muscle can result in a specific pathological syndrome characterized by massive release of myoglobin, known as:
(A) High-altitude pulmonary brisket disease
(B) Hypoxic subacute high-altitude disease
(C) Compartment syndrome / Crush syndrome
(D) Localized Type III Arthus reaction

2001

139. Which of the following is a classic pathological outcome of massive myoglobin release secondary to severe muscle contusions?
(A) Direct, immediate development of congestive heart failure
(B) Permanent, widespread depigmentation of the epidermis
(C) Photochemical formation of pyrimidine dimers in the liver
(D) Acute renal failure due to myoglobinuric nephrosis

2022

140. A sharp-edged cut whose depth is significantly greater than its surface length is most accurately classified as a:
(A) Puncture
(B) Contusion
(C) Abrasion
(D) Superficial laceration

2024

141. The immediate cause of death in massive, acute heat stroke is most frequently:
(A) Complete and sudden destruction of circulating red blood cells
(B) Cardiovascular collapse from vasodilation and hemoconcentration
(C) Sudden, life-threatening hypocalcemia and electrolyte imbalance
(D) Instant, irreversible thermal coagulation of the entire brain stem

2016

142. The pathological hallmark of trench foot (a severe non-freezing cold injury) involves:
(A) Complete, solid freezing of intracellular fluids and rupture of cell membranes
(B) Prolonged vasoconstriction leading to edema, endothelial damage, and blistering
(C) Immediate evaporation of joint fluid and sudden crystallization of bone salts
(D) Rapid, systemic accumulation of phylloerythrin within the affected extremity

2017

143. Which environmental condition vastly accelerates the onset of clinical hypothermia in animals exposed to low temperatures?
(A) High ambient humidity and strong winds
(B) Excessive dietary salt and water intake
(C) Very thick, dense protective undercoats
(D) Markedly dark hair or skin pigmentation

2019

144. Hypoxia specifically refers to:
(A) Total and absolute absence of oxygen in metabolizing tissues
(B) Abnormally high levels of carbon dioxide within the blood
(C) Reduced availability of oxygen to active cells and tissues
(D) Complete mechanical inability of the heart

Downloaded from www.pdf.tube

to pump any blood

2024

145. At high altitudes, the body attempts to compensate for chronic hypoxia primarily by:
(A) Significantly decreasing the systemic respiratory rate
(B) Markedly decreasing the resting myocardial heart rate
(C) Progressively shrinking the overall volume of the lungs
(D) Increasing the production of oxygen-carrying red blood cells

2015

146. A photodynamic agent structurally interacts with UV light to directly produce which damaging species within the skin?
(A) Active components of the complement MAC
(B) Systemic radioactive heavy metal isotopes
(C) Reactive oxygen species and free radicals
(D) Localized vascular fibrinoid necrosis

2013

147. Which breed characteristic acts as a major intrinsic predisposing factor for squamous cell carcinoma of the eye in cattle?
(A) Uniformly solid black coat color
(B) Lack of circumocular pigmentation
(C) Possession of homozygous polled genetics
(D) Exceedingly large and heavy horn size

2002

148. Identify the most critical defense mechanism mammals possess against the penetrative and DNA-damaging effects of natural non-ionizing UV radiation.
(A) Production of an exceptionally thick subcutaneous fat layer
(B) Rapid, shallow panting to cool down the outer skin surface
(C) The continuous, rapid shedding of the outer epidermal layers
(D) Epidermal melanin synthesis and distribution by melanocytes

2023

149. Gamma rays cause more extensive deep tissue injury than standard UV light primarily because they:
(A) Possess high energy and penetrance, ionizing deep intracellular molecules
(B) Are significantly less energetic than standard non-ionizing ultraviolet light
(C) Exclusively target and damage the highly superficial epidermal cell layers
(D) Selectively damage or degrade melanin pigment molecules within the dermis

2005

150. A major late-stage, chronic pathological consequence of surviving acute exposure to ionizing radiation is:
(A) Sudden, uncontrolled overgrowth of all localized hair follicles
(B) Immediate and significant weight gain due to metabolic changes
(C) Development of secondary malignancies due to permanent DNA mutations
(D) Complete, sudden reversal of all previously inherited genetic traits

2019

151. Which factors determine the severity of injury sustained from an electric shock?
(A) The voltage, current type, tissue resistance, and pathway of the current
(B) The exact ambient temperature and humidity of the surrounding environment
(C) The physiological age, biological sex, and current weight of the animal
(D) The specific coat color, hair length, and skin thickness of the subject

NODIA

2019

152. Tissues with the lowest electrical resistance suffer the most immediate damage during electrocution. Which of the following has the lowest electrical resistance?
(A) Dry, heavily keratinized skin
(B) Bone
(C) Dense fat
(D) Blood and nerve tissues

2005

153. What is the fundamental difference between exogenous inorganic and exogenous organic chemical agents?
(A) Inorganic agents are strictly synthesized by animals, whereas organic agents are only produced by plants.
(B) Inorganic agents lack carbon-hydrogen bonds, whereas organic agents are complex carbon-based compounds.
(C) Inorganic agents are strictly inhaled into lungs, while organic agents are exclusively injected into veins.
(D) Inorganic agents are universally harmless to hosts, while organic agents are always immediately fatal.

2023
154. Chronic lead toxicity is uniquely characterized pathologically by:
(A) The presence of pathognomonic crystalline structures and cellular casts within the spleen
(B) The sudden, unexpected appearance of multiple massive adenomas within the liver cortex
(C) Basophilic stippling of erythrocytes and intranuclear inclusions in renal tubules
(D) Total and irreversible loss of myelin throughout the entire central nervous system

2008
155. Arsenic toxicity is primarily known for causing catastrophic damage to:
(A) The development and structural integrity of the skull bones
(B) The physiological function of the retinal pigment epithelium
(C) The continuous mineralization and formation of tooth enamel
(D) The systemic microvasculature, causing hemorrhage and edema

2005
156. Iatrogenic hyperadrenocorticism (Cushing's syndrome) is a classic example of iatrogenic toxicity caused by the prolonged administration of:
(A) Glucocorticoids (steroids)
(B) Penicillin G (antibiotics)
(C) Polyvalent viral vaccines
(D) Physiological normal saline

2012
157. Which type of feed adulterant causes catastrophic renal failure in dogs and cats by forming insoluble crystals in the renal tubules?
(A) Copper sulfate
(B) Melamine and cyanuric acid
(C) Aflatoxin
(D) High-fructose corn syrup

2014
158. Endogenous chemical injury can result from severe hepatic failure, where the accumulation of toxic ammonia in the blood leads to a brain-damaging syndrome known as:
(A) Uremic stomatitis
(B) Hepatoencephalopathy
(C) Congenital porphyria
(D) Hypoxia

2019
159. In uremia, the high concentration of circulating urea leads directly to mucosal ulceration primarily because:
(A) Salivary and gastric bacteria convert urea into caustic ammonia, severely burning the mucosa.
(B) Urea forms sharp macroscopic crystals that mechanically cut the mucosa.
(C) Urea aggressively dissolves tooth enamel, exposing the gums.
(D) Urea acts as a powerful localized vasoconstrictor, stopping all blood flow to the mouth.

2012
160. Ketoacidosis significantly depresses blood pH. This severe metabolic acidosis directly disrupts cellular function by:
(A) Spontaneously generating extreme internal metabolic heat
(B) Blocking all cellular ultraviolet radiation absorption
(C) Denaturing systemic proteins and impairing enzymatic activity
(D) Rapidly turning circulating blood into solid vascular clots

2009
161. Post-hepatic icterus is most likely to be diagnosed in an animal suffering from:
(A) A severe autoimmune attack on circulating red blood cells
(B) A massive chemical overdose of toxic heavy metal compounds
(C) A congenital, severe metabolic deficiency of dietary iron
(D) A complete mechanical blockage of the common bile duct

2021
162. Blood transfusion reactions, where pre-existing host antibodies aggressively attack mismatched donor red blood cells, are mediated by:
(A) Type I hypersensitivity
(B) Type II hypersensitivity
(C) Type III hypersensitivity
(D) Type IV hypersensitivity

2014
163. In Type II hypersensitivity, opsonization refers to the process where:
(A) Recruited neutrophils die rapidly and form localized pus
(B) Target cells are antibody-coated to enhance phagocytosis

Click Here to Buy book on Flipkart

(C) Activated mast cells release massive amounts of histamine
(D) Generated free radicals punch holes in the target cell membrane

2004
164. The primary antibody classes involved in activating the complement cascade during Type II and Type III hypersensitivity reactions are:
(A) IgE and IgD
(B) IgA and IgE
(C) IgA and IgG
(D) IgG and IgM

2010
165. Which pathological lesion is an absolute hallmark of the Arthus reaction (localized Type III hypersensitivity)?
(A) Severe acute necrotizing vasculitis with local hemorrhage and thrombosis
(B) The formation of large, organized caseous granulomas
(C) The development of widespread squamous cell carcinomas
(D) The complete, clean dissolution of the affected tissue without inflammation

2018
166. Immune complexes deposited in the renal glomeruli cause severe damage primarily because they:
(A) Act as physical sharp microcrystals that directly shred the basement membrane
(B) Directly inhibit the physiological sodium-potassium pump of adjacent podocytes
(C) Activate complement to recruit neutrophils that release destructive proteases and ROS
(D) Induce immediate, programmed cell death (apoptosis) in all surrounding cells

2006
167. During lipid peroxidation, the initiation phase is characterized by which of the following events?
(A) The enzymatic conversion of lipid hydroperoxides to alcohols by glutathione peroxidase
(B) The abstraction of a hydrogen atom from a polyunsaturated fatty acid by a reactive free radical
(C) The non-enzymatic conjugation of malondialdehyde with cellular proteins
(D) The donation of an electron from Vitamin E to a lipid peroxyl radical

2018
168. The destructive cascade where free radicals react with membrane lipids, creating lipid radicals that continuously react with adjacent lipids, is called:
(A) The Fenton chain reaction
(B) The Haber-Weiss loop
(C) Protein cross-linking
(D) Lipid peroxidation propagation

2023
169. During the respiratory burst in neutrophils, myeloperoxidase (MPO) utilizes hydrogen peroxide and halide ions to produce which highly bactericidal substance?
(A) Hydroxyl radical
(B) Hypochlorous acid (HOCl)
(C) Peroxynitrite
(D) Singlet oxygen

2017
170. Which vital cellular structure is most immediately structurally compromised by active lipid peroxidation?
(A) The protein-rich nucleolus within the nucleus
(B) The sliding actin-myosin contractile complex
(C) Phospholipid bilayers of cell and organelle membranes
(D) The protein-synthesizing ribosomal machinery

2014
171. The Haber-Weiss reaction generates highly reactive hydroxyl radicals from:
(A) Water and carbon dioxide
(B) Melanin and ultraviolet light
(C) Urea and ammonia
(D) Superoxide and hydrogen peroxide

2002
172. When intracellular reactive oxygen species (ROS) levels overwhelm antioxidant defenses and cause severe, irreparable damage, the cell typically:
(A) Undergoes programmed cell death (apoptosis) or necrosis
(B) Permanently ceases metabolic activity while remaining structurally intact
(C) Intentionally mutates its nuclear genome to adapt to the oxidative environment
(D) Converts to a fully anaerobic photosynthetic metabolic pathway

2023
173. What is the fundamental role of the endogenous cellular antioxidant system?
(A) To generate massive amounts of free radicals

Downloaded from www.pdf.tube

to destroy bacteria
(B) To safely donate electrons to free radicals, neutralizing them
(C) To form permanent, stable covalent cross-links between proteins
(D) To significantly increase the intracellular concentration of iron

2014
174. Which of the following plasma proteins serves as an important antioxidant defense by binding free iron, thereby preventing it from participating in the Fenton reaction?
(A) Albumin
(B) Transferrin
(C) Ceruloplasmin
(D) Haptoglobin

2021
175. Which enzyme is responsible for regenerating reduced glutathione (GSH) from oxidized glutathione (GSSG), utilizing NADPH as a reducing equivalent?
(A) Glutathione S-transferase
(B) Glutathione reductase
(C) Glucose-6-phosphate dehydrogenase
(D) Glutamyl transpeptidase

2017
176. Glutathione peroxidase reduces hydrogen peroxide to water by oxidizing which endogenous tripeptide cofactor?
(A) Methionine
(B) Reduced glutathione (GSH)
(C) Metallothionein
(D) Ubiquinone

2004
177. Prolonged, unopposed generation of Reactive Oxygen Species (ROS) has been heavily implicated in the pathogenesis of all the following EXCEPT:
(A) Cellular aging
(B) Ischemia-reperfusion injury
(C) Chemical and radiation-induced carcinogenesis
(D) Normal, healthy mitosis

2025
178. Cellular swelling, a hallmark of early, reversible cell injury resulting from any fundamental stressor, is primarily caused by:
(A) The mass synthesis and intracellular accumulation of neutral lipids
(B) The rapid, completely uncontrolled replication of the nucleus
(C) Failure of the Na^+/K^+ ATPase pump, causing sodium and water influx
(D) The sudden, pathological enlargement of the cellular Golgi apparatus

2014
179. A morphological change characterizing irreversible cell death, in which the nucleus violently fragments into small, dense, basophilic particles, is called:
(A) Karyolysis
(B) Karyorrhexis
(C) Pyknosis
(D) Plasmolysis

2003
180. The shrinkage and extreme condensation of a dead cell's nucleus into a small, dark, structureless mass is known as:
(A) Karyorrhexis
(B) Karyolysis
(C) Apoptosis
(D) Pyknosis

2021
181. Coagulative necrosis, commonly caused by severe ischemia or extreme heat, is characterized microscopically by:
(A) Preservation of cell outlines and tissue architecture despite cell death
(B) Complete and immediate dissolution of cellular architecture into a liquid
(C) The rapid formation of massive, multinucleated cellular syncytia
(D) The sudden, widespread formation of large intracellular lipid vacuoles

NODIA

2024
182. Which type of necrosis is uniquely characterized by the complete digestion of dead cells into a viscous, liquid mass, typical of severe bacterial infections and brain infarcts?
(A) Coagulative necrosis
(B) Caseous necrosis
(C) Liquefactive necrosis
(D) Fat necrosis

2004
183. Caseous necrosis, historically associated with infections like Tuberculosis, is grossly described as appearing:
(A) Dry, friable, and "cheese-like"
(B) Clear and fluid-filled
(C) Rock hard and strictly composed of bone
(D) Black and severely putrefied

2006

184. Pathological calcification occurring in dead or dying tissues, despite normal serum calcium levels, is defined as:
(A) Metastatic calcification
(B) Idiopathic calcification
(C) Iatrogenic calcification
(D) Dystrophic calcification

2002

185. Metastatic calcification occurs primarily due to:
(A) Direct, acute trauma localized to a single skeletal muscle group
(B) Persistent hypercalcemia, depositing calcium in normal tissues
(C) Normal serum calcium levels acting on severely necrotic tissues
(D) The massive, uncontrolled overproduction of active Vitamin C

2025

186. Which underlying condition is most likely to lead to metastatic calcification?
(A) A localized, healing surgical incision in the skin
(B) An old, chronic encapsulated parasitic cyst in tissue
(C) Chronic renal failure causing secondary hyperparathyroidism
(D) A singular, acute mechanical contusion on the animal's flank

2007

187. The term applied to necrotic tissue that has been secondarily invaded and digested by putrefactive saprophytic bacteria is:
(A) Infarction
(B) Apoptosis
(C) Ulceration
(D) Gangrene

2007

188. Dry gangrene frequently occurs on the extremities (e.g., ears, tail) primarily as a consequence of:
(A) Chronic ischemia causing coagulative necrosis and desiccation
(B) Severe, acute systemic bacterial septicemia and endotoxemia
(C) Massive, systemic overhydration and cellular fluid overload
(D) An acute, severe localized Type I hypersensitivity reaction

2008

189. Apoptosis is best defined as:
(A) Accidental, passive cell death that triggers a massive local inflammation
(B) Programmed, energy-dependent cell death without surrounding inflammation
(C) The traumatic, mechanical bursting of a cell due to direct physical impact
(D) The rapid, ischemic death of a massive swath of tissue from a blocked artery

2019

190. Which structural event is a classic feature of an apoptotic cell?
(A) Massive, rapid swelling of the cell followed by membrane rupture
(B) Formation of apoptotic bodies, which are phagocytosed by adjacent cells
(C) The immediate leakage of active lysosomal enzymes into the outer space
(D) Complete, sudden liquefaction of the surrounding extracellular matrix

2024

191. Accumulation of normal cellular constituents or abnormal substances is a hallmark of metabolic derangement. Steatosis (fatty change) most frequently occurs in which organ due to its central role in fat metabolism?
(A) The liver
(B) The brain
(C) The lungs
(D) The spleen

2007

192. Which of the following best describes amyloidosis?
(A) Massive accumulation of normal glycogen within renal tubular cells
(B) The excessive intracellular storage of iron within liver macrophages
(C) Extracellular accumulation of insoluble, misfolded fibrillar proteins
(D) The viral-mediated conversion of normal host DNA into foreign viral RNA

2022

193. Grossly, tissues heavily affected by severe amyloidosis typically appear:
(A) Bright red and highly vascular
(B) Severely shrunken and black
(C) Completely dissolved into a liquid state
(D) Enlarged, pale, firm, and waxy

2003

194. Melanin is a dark endogenous pigment primarily responsible for protecting the skin against UV radiation. The pathological lack of melanin production due to a genetic enzyme defect is known as:

Click Here to Buy book on Amazon

(A) Melanoma
(B) Porphyria
(C) Albinism
(D) Icterus

2002
195. Hemosiderin is a golden-yellow to brown granular intracellular pigment that represents:
(A) Accumulated, partially digested cellular lipid and protein residues
(B) Intracellular storage of iron derived from hemoglobin breakdown
(C) Pathological accumulation of undigested bile pigments in hepatocytes
(D) Massive, abnormal extracellular deposits of active calcium crystals

2008
196. The "wear-and-tear" pigment, consisting of lipid-protein complexes that accumulate intracellularly in aging post-mitotic cells (like neurons and cardiac myocytes), is:
(A) Melanin
(B) Bilirubin
(C) Porphyrin
(D) Lipofuscin

2009
197. Anthracosis, a common incidental finding in animals living in polluted urban environments, involves the accumulation of which exogenous substance in alveolar macrophages?
(A) Carbon particles
(B) Silica dust
(C) Asbestos fibers
(D) Iron dust

2019
198. The specific localized area of ischemic necrosis within a tissue or organ, caused by occlusion of either its arterial supply or its venous drainage, is definitively termed an:
(A) Abscess
(B) Ulcer
(C) Infarct
(D) Embolus

2024
199. A detached, intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin is called an:
(A) Embolus
(B) Infarct
(C) Aneurysm
(D) Atheroma

NODIA

2001
200. Hyperemia is defined as:
(A) A passive process resulting from impaired venous outflow from a tissue
(B) The absolute cessation of all blood flow to a tissue
(C) The bleeding of blood directly into a body cavity
(D) An active process characterized by increased arterial blood flow to a tissue

2014
201. Congestion (passive hyperemia) fundamentally differs from active hyperemia because it involves:
(A) Increased active arterial inflow, making the affected tissue appear bright red
(B) Decreased venous outflow, causing hypoxia, cyanosis, and dark discoloration
(C) The massive, immediate rupture of all surrounding capillary blood vessels
(D) Selective removal of oxygen-carrying red cells from the vascular space

2002
202. Chronic passive congestion of the liver, typically secondary to right-sided heart failure, classically produces a macroscopic patterned appearance known as:
(A) Gunmetal liver
(B) Fibrinous liver
(C) Nutmeg liver
(D) Cirrhotic liver

2001
203. The abnormal accumulation of excess fluid within the interstitial tissue spaces or body cavities is known as:
(A) Hemorrhage
(B) Embolism
(C) Ischemia
(D) Edema

2014
204. Which of the following is a primary pathophysiological mechanism that directly causes the formation of edema?
(A) Increased capillary endothelial permeability
(B) Increased systemic plasma oncotic pressure
(C) Decreased capillary hydrostatic pressure
(D) Increased physiological lymphatic drainage

2011
205. Hypoproteinemia (e.g., from severe parasitism or starvation) consistently leads to systemic edema through which mechanism?
(A) Massive, acute increases in vascular

hydrostatic pressure
(B) A profound decrease in intravascular colloid osmotic pressure
(C) Total, irreversible obstruction of the regional lymphatic system
(D) Immediate, complete destruction of the capillary basement membrane

2001

206. Severe generalized edema, characterized by profound fluid accumulation in subcutaneous tissues throughout the entire body, is specifically termed:
(A) Ascites
(B) Anasarca
(C) Hydrothorax
(D) Hydropericardium

2015

207. Exudate is fundamentally distinguished from transudate by being an inflammatory fluid that is:
(A) Highly protein-rich with a high specific gravity and numerous cells
(B) Low in protein with a low specific gravity and very few cells
(C) Exclusively composed of pure, clear water
(D) Formed strictly due to heart failure without any vascular damage

2006

208. Hemorrhage refers to the escape of blood from the cardiovascular system. Minute, pinpoint (1-2 mm) hemorrhages on skin or mucosal surfaces are specifically termed:
(A) Ecchymoses
(B) Purpura
(C) Petechiae
(D) Hematomas

2006

209. A localized, massive accumulation of extravasated blood within a tissue space, often forming a palpable swelling or clot, is called a:
(A) Petechia
(B) Purpura
(C) Contusion
(D) Hematoma

2011

210. The pathological process consisting of a cascade of events leading to the formation of a solid mass of blood constituents within the unbroken cardiovascular system of a living animal is called:
(A) Embolism
(B) Infarction
(C) Thrombosis
(D) Ischemia

****

Downloaded from www.pdf.tube

ANSWERS

1. (A) General pathology
General pathology focuses on the basic mechanisms of cellular and tissue injury, establishing core principles such as necrosis, apoptosis, inflammation, and neoplasia. This foundational branch underpins all specialized areas of veterinary pathology by explaining how cells react to injurious stimuli, regardless of the specific organ system or animal species involved in the disease process.

2. (C) Systemic pathology
Systemic pathology examines the specific diseases, alterations, and lesions occurring within particular organs and anatomical systems, such as the respiratory, cardiovascular, urinary, or digestive systems. It builds upon general pathological principles to study how specific tissues respond to unique insults, allowing clinicians and pathologists to understand localized manifestations of diverse disease processes in veterinary patients.

3. (A) It analyzes blood, bodily fluids, and cytology to diagnose disease.
Clinical pathology focuses on the diagnostic analysis of blood, urine, cerebrospinal fluid, joint fluid, and cytological aspirates from living animals. By utilizing hematology, clinical chemistry, urinalysis, and microscopic cell evaluation, this branch provides vital information regarding organ function, endocrine status, hematological health, and cytological abnormalities to guide therapeutic decisions and establish accurate clinical prognoses.

4. (D) Molecular pathology is limited to the visual inspection of external lesions.
Molecular pathology utilizes advanced subcellular and genetic techniques to identify mutations, nucleic acid sequences, and protein alterations in diseased tissues, rather than being restricted to simple visual or macroscopical inspection. This specialized field combines molecular biology with morphological pathology to provide high-resolution diagnostic insights, characterization of infectious agents, and detection of hereditary genetic disorders directly.

5. (A) P-1, Q-2, R-3, S-4
Pathology branches are distinct in their diagnostic applications and materials. Surgical biopsy examines excised living tissue, clinical chemistry analyzes blood and body fluids, molecular testing targets DNA or RNA sequences, and anatomic pathology performs post-mortem necropsies or gross organ assessments. Understanding these specific diagnostic mediums is essential for selecting appropriate diagnostic tests in veterinary medicine.

6. (C) Rudolf Virchow
Rudolf Virchow is recognized as the pioneer of cellular pathology because he demonstrated that all diseases originate as alterations within individual cells. By showing that cells arise only from pre-existing cells, he shifted the paradigm of medicine away from ancient humoral theories and established the modern scientific foundation of cellular injury within global veterinary science.

7. (D) Giovanni Morgagni
Giovanni Morgagni laid the foundation for pathological anatomy by establishing that organs are the primary anatomical sites of disease. Through his extensive post-mortem examinations, he meticulously correlated clinical symptoms observed during a patient's life with structural changes found within specific organs after death, effectively transitioning medical science into an era of organ-based clinical-pathological correlation.

8. (A) John Hunter
John Hunter was a pioneering British surgeon and biologist who heavily utilized experimental pathology to investigate the physiological mechanisms of tissue repair, bone healing, and inflammatory responses. His innovative, evidence-based approach to wound healing and vascular biology introduced rigorous scientific experimentation to the study of surgical disease, greatly advancing surgical techniques and active tissue regeneration.

NODIA

9. (B) The formulation of postulates linking pathogens to diseases
Robert Koch made a monumental contribution to infectious disease pathology by formulating Koch's postulates. These scientific criteria established a standardized, logical framework for proving that a specific micro-organism is the causative agent of a particular infectious disease. His rigorous methodology revolutionized the field of microbiology, allowing definitive identification of pathogens responsible for various infectious diseases.

10. (B) Rudolf Virchow
The biological concept "omnis cellula e cellula," meaning that every living cell arises only from a pre-existing cell, was popularized and championed by Rudolf Virchow. This core tenet of cell theory successfully refuted ancient ideas of spontaneous generation, establishing that tissue growth, physiological repair, and pathological neoplasms develop through the continuous division of existing cells.

11. (C) A is false but R is true.
Giovanni Morgagni is renowned for correlating clinical symptoms observed during life with gross anatomical changes discovered during post-mortem autopsies. However, he did not formulate the germ theory of disease, which was developed much later by scientists like Louis Pasteur and Robert Koch. Therefore, the assertion is false while the supporting reason is historically completely correct.

12. (C) Refinements in light microscopy and tissue staining techniques
Modern diagnostic histopathology arose directly due to significant advancements in light microscopes and the development of specialized chemical staining techniques. Techniques like hematoxylin and eosin staining allowed pathologists to visualize fine cellular structures and distinguish healthy tissues from diseased morphology, transitioning diagnostic pathology from macroscopical necropsies to precise microscopic tissue analyses and evaluations.

13. (D) Lesion
A lesion is defined as any structural, physical, or functional deviation from normal tissue morphology resulting from a disease process or physical trauma. Pathologists identify, describe, and analyze these lesions macroscopically and microscopically during biopsies and post-mortem examinations to determine the underlying cause, progression, severity, and definitive classification of various veterinary diseases.

14. (C) Pathogenesis
Pathogenesis is the step-by-step sequence of events and mechanisms that occur from the initial contact with an etiological agent to the final functional and structural manifestation of a disease. It describes how cellular injury, biochemical alterations, tissue responses, and vascular changes progress over time, providing a comprehensive timeline of veterinary disease development processes.

15. (B) Pathognomonic
A pathognomonic lesion is a highly specific, unique structural alteration that occurs exclusively in one particular disease, allowing for an immediate, definitive diagnosis. Identifying such a characteristic finding provides absolute diagnostic certainty without the necessity of further laboratory confirmation, as no other disease or insult can produce the identical tissue changes.

16. (D) Necropsy is a post-mortem exam, while biopsy evaluates tissue from a living patient.
A necropsy, or autopsy, is a comprehensive post-mortem examination performed on a deceased animal carcass to investigate the cause of death and characterize systemic lesions. In contrast, a biopsy is the surgical excision of a small tissue sample from a living animal, enabling diagnostic evaluation and pathological grading of lesions while the patient survives.

17. (A) I and III
Artifacts represent artificial alterations introduced into tissues during post-mortem decomposition, surgical removal, tissue fixation, embedding, sectioning, or staining processes. Because they do not represent true antemortem lesions, pathologists must remain highly skilled at identifying artifacts to avoid misinterpreting these non-specific technical errors as genuine pathognomonic features or diagnostic indicators of an active disease.

18. (C) Sign
A clinical sign is an objective, observable, and measurable manifestation of disease in an animal, detected during a physical examination or

Click Here to Buy book on Flipkart

diagnostic assessment by a veterinarian. Unlike symptoms, which are subjective sensations experienced exclusively by the patient, clinical signs include measurable indicators like elevated body temperature, altered heart rate, or visible lesions.

19. (A) Symptom
A symptom represents a subjective sensation or physical discomfort that only the patient can feel and describe, such as nausea, localized pain, or dizziness. Because veterinary patients cannot verbally communicate these internal sensations, identifying true symptoms in animals is highly challenging, making clinicians heavily reliant on objective, observable clinical signs and tests.

20. (D) Syndrome
A syndrome is a distinct collection of multiple concurrent clinical signs, symptoms, or pathological lesions that consistently occur together to characterize a specific disease or abnormal state. Although the precise underlying etiology of a syndrome might initially remain unknown, this clustered clinical presentation provides a highly recognizable, patterns-based diagnostic profile for veterinary clinicians.

21. (B) Diagnosis
A diagnosis is the formal scientific determination and identification of the nature, cause, and extent of a disease. It is established through a systematic integration of clinical history, physical signs, morphologic lesions, and diagnostic testing results, allowing veterinarians to differentiate between similar conditions, select appropriate therapies, and implement highly targeted control measures.

22. (C) Prognosis
A prognosis is a professional medical forecast predicting the most probable course, duration, clinical outcome, and chances of recovery for an animal suffering from a specific disease. This prediction is formulated by carefully evaluating the severity of lesions, the standard pathogenesis of the condition, and availability of effective therapeutic clinical interventions.

23. (D) Exposure, incubation period, clinical signs, structural alterations
The progression of an infectious disease typically begins with the host's initial exposure to a pathogen, followed by the incubation period where the pathogen replicates. Next, the animal develops observable clinical signs as cell injury intensifies, eventually leading to permanent or temporary structural alterations within the affected tissues that can be observed macroscopically or microscopically.

24. (A) Morbidity
Morbidity represents the ratio or proportion of animals within a specific population that contract a particular disease over a defined period of time. It acts as an essential epidemiological metric for assessing the transmissibility, infectivity, and general prevalence of a disease within a herd, flock, or regional animal population, independent of fatal disease outcomes.

25. (B) Mortality
Mortality is an epidemiological term defined as the ratio or number of deaths caused by a specific disease relative to the total population of susceptible animals over a set timeframe. Monitoring mortality rates is critical for evaluating the virulence, lethality, and clinical severity of pathogenic outbreaks, as well as assessing the efficacy of preventive measures.

NODIA

26. (B) Incubation period
The incubation period is the precise time interval spanning from the host's initial exposure to a pathogenic infectious agent up to the very first manifestation of clinical signs. During this silent phase, the pathogen actively replicates, colonizes target organs, and initiates cellular damage, which must reach a critical threshold before clinical signs become observable.

27. (A) Animate causes
Animate causes of disease comprise living organisms and replication-competent biological agents, including viruses, bacteria, protozoa, fungi, helminths, and arthropod parasites. These biological entities actively invade, multiply within, and damage host tissues through diverse mechanisms like toxin production, direct cellular lysis, or the induction of damaging host immune and inflammatory cellular responses.

28. (C) Inanimate cause
Inanimate causes of disease represent non-living agents and environmental factors that induce pathology, which are broadly grouped into physical, chemical, and nutritional factors. Nutritional deficiencies, extreme temperature

exposure, physical trauma, and heavy metal toxicities are all classified as inanimate insults because they do not involve replication-competent biological pathogens or living micro-organisms.

29. (A) I, II and IV
Acquired diseases develop after birth due to external environmental factors, not from inherited genes. Congenital diseases are present at birth but can stem from intrauterine environmental factors, such as maternal toxins or infections, rather than purely genetic mutations. Additionally, physical agents like extreme cold and radiation are inanimate, not animate, etiologies.

30. (C) Acquired
Acquired diseases are those that develop in an animal at any stage of life after its birth. These conditions are triggered by external factors, including microbial infections, environmental stressors, chemical toxicities, physical trauma, or nutritional imbalances, contrasting directly with congenital or genetic diseases that are present at or before the actual birth.

31. (B) Congenital
Congenital anomalies or defects are anatomical, structural, or functional abnormalities that are present in an animal precisely at the time of its birth. These conditions can arise from inherited genetic mutations, chromosomal aberrations, or adverse intrauterine environmental impacts, such as maternal exposure to teratogenic toxins, infectious agents, or deficiencies during early fetal development.

32. (D) Hip dysplasia driven by breed genetics
Hip dysplasia is a structural, degenerative orthopedic disease primarily driven by intrinsic genetic predisposing factors associated with specific dog breeds, especially large and giant breeds. Unlike extrinsic environmental insults such as frostbite, toxicities, or nutritional deficiencies, this condition is determined by inherited genetic susceptibility that alters joint development and leads to progressive skeletal laxity.

33. (A) Idiosyncrasy
An idiosyncrasy is an abnormal, highly unpredictable individual hypersusceptibility or adverse reaction to a drug, chemical, or therapeutic agent. This reaction is completely unrelated to species or breed-wide norms and is not dose-dependent, representing a genetically determined, unique metabolic or immunological response that differs fundamentally from the standard pharmacological effects of the compound.

34. (C) Intrinsic genetic factors
The high incidence of melanomas in grey horses represents an intrinsic genetic predisposing factor. The specific genes responsible for the progressive greying of the hair coat are biologically linked to altered melanocyte metabolism and dermal melanin accumulation, creating an inherited, internal susceptibility to neoplastic transformation of pigment cells that is independent of external stressors.

35. (C) P-3, Q-4, R-2, S-1
Intrinsic predisposing factors are internal biological traits of the host. Species determines viral susceptibility, as seen with canine distemper affecting dogs but not cattle. Breed influences conformation, such as brachycephalic airway syndrome in Pugs. Age drives degenerative conditions like osteoarthritis in geriatric animals, while sex dictates hormone-dependent pathologies like prostatic hyperplasia in males.

NODIA

36. (D) Climatic stress from extreme weather
Extrinsic predisposing factors originate entirely from outside the animal's body. Climatic stress, such as exposure to extreme heat, cold, or high humidity, acts as an environmental trigger that suppresses immune function and alters physiological homeostasis, thereby increasing the host's vulnerability to secondary infections or metabolic disturbances, unlike genetic, sex-linked, or breed-conformation factors.

37. (B) Starvation
Starvation is a severe nutritional condition that develops due to a prolonged, inadequate intake of dietary energy and protein. This systemic energy deficit forces the animal's body to catabolize stored adipose tissue and skeletal muscle proteins to maintain baseline physiological functions, resulting in progressive muscle wasting, severe cachexia, and immunologic suppression.

38. (C) Managemental faults
Poor ventilation, inadequate housing, and severe overcrowding in livestock facilities represent

critical managemental faults that serve as major extrinsic predisposing factors. These human-controlled environmental deficiencies directly compromise the health of animals by increasing air pollutants like ammonia, raising relative humidity, and facilitating the rapid, horizontal transmission of respiratory and gastrointestinal pathogens.

39. (D) By increasing stress and pathogen exposure
Overcrowding and managemental faults act as profound stressors that stimulate the hypothalamic-pituitary-adrenal axis, releasing endogenous cortisol. Cortisol suppresses immunologic responses, decreasing host defense mechanisms. Simultaneously, close confinement increases animal-to-pathogen exposure and facilitates rapid pathogen transmission, dramatically raising the incidence of clinical disease outbreaks within the herd or flock of livestock animals.

40. (A) Abrasion
An abrasion is a superficial mechanical injury restricted to the epidermis, caused by friction from rubbing or scraping against a rough, hard surface. This type of trauma leaves the underlying dermis largely intact, resulting in minimal blood loss, although the exposed raw surface can leak serous fluid and predispose the area to infection.

41. (A) I, II, IV and V
Mechanical injuries are defined by structural tissue damage patterns. A contusion involves intact skin with ruptured deep vessels, an incision represents a sharp clean cut, punctures are narrow deep penetrations, and fractures disrupt bone continuity. Abrasions are superficial epithelial scrapes, meaning the statement describing them as deep, irregular tissue tears is histologically incorrect.

42. (B) Laceration
A laceration is a mechanical injury characterized by an irregular, jagged tear in the skin and underlying tissues, caused by blunt shearing forces or ripping trauma. Unlike incisions, lacerations typically feature crushed wound margins, damaged blood vessels, and bridging tissue strands within the wound bed, which often complicates primary surgical tissue healing times.

43. (A) Incision
An incision is a clean, linear wound with smooth margins, produced by a sharp-edged instrument like a surgical scalpel. Because the surrounding tissues experience minimal crushing or devitalization, the vascular integrity is spared up to the cut margins, allowing for precise alignment, minimal granulation tissue formation, and rapid tissue healing.

44. (C) Puncture
A puncture is a deep, narrow wound caused by a sharp, pointed object, such as a nail, tooth, or wire, which penetrates deep tissue layers. The narrow entrance wound restricts airflow, creating an optimal anaerobic microenvironment within the damaged tissue that heavily favors the proliferation of anaerobic bacterial pathogens like Clostridium tetani.

45. (D) Fracture
A fracture is defined as the partial or complete discontinuity of bone or cartilage structure, typically resulting from excessive mechanical force, pathological bone weakening, or repetitive stress. The healing of a fracture requires temporary stabilization to facilitate the formation of a fibrocartilaginous callus, which gradually mineralizes and remodels back into organized lamellar bone.

46. (C) Full-thickness destruction of skin and local nerves
A third-degree thermal burn is characterized by full-thickness destruction of both the epidermis and the dermis, often extending into deeper subcutaneous tissues, muscles, or bone. Because the local sensory nerve endings residing within the dermis are completely destroyed, these severe lesions are clinically anesthetic, meaning the animal feels no pain at the burn site.

47. (C) II, IV, I, III
The pathogenesis of heat stroke begins with prolonged exposure to high ambient temperatures and humidity. As environmental stressors exceed the host's capacity, thermoregulatory mechanisms fail, causing severe hyperthermia. This profound core body temperature elevation triggers extensive systemic protein denaturation, cell membrane injury, and intravascular microthrombosis, rapidly culminating in multi-organ dysfunction syndrome and death.

Downloaded from www.pdf.tube

48. (D) Profound peripheral vasodilation leading to circulatory shock
Heat exhaustion is a clinical syndrome of thermoregulatory strain characterized by profound peripheral vasodilation as the body attempts to dissipate excess heat. This vascular expansion, combined with dehydration and fluid loss from sweating or panting, leads to decreased venous return, diminished cardiac output, and acute circulatory shock, before protein denaturation occurs.

49. (A) Protein denaturation and coagulation
At the cellular level, the fundamental mechanism of local tissue injury from exposure to extreme heat is the rapid, irreversible denaturation and coagulation of structural and enzymatic proteins. This thermal disruption destroys cellular membrane integrity, inactivates essential metabolic enzymes, and precipitates cytoplasmic proteins, resulting in immediate coagulative necrosis of affected tissue.

50. (C) Erythema and mild pain
First-degree burns are superficial thermal injuries limited entirely to the epidermal layer of the skin. They are clinically characterized by active hyperemia, which presents as visible erythema, accompanied by localized edema and mild pain due to the stimulation of intact epidermal nociceptors, without any vesicle formation, scarring, or deep tissue necrosis processes.

51. (A) Ice crystal formation and thrombosis-induced ischemia
Frostbite-induced local tissue injury is caused by a dual mechanism. Initially, severe cold causes extracellular and intracellular ice crystal formation, which physically disrupts cell membranes and de-vitalizes cells. Subsequently, intense localized vasoconstriction is followed by endothelial injury, platelet aggregation, and intravascular thrombosis, resulting in severe tissue ischemia and localized dry gangrene.

52. (D) Hypothermia
Hypothermia is a critical, life-threatening clinical state characterized by an abnormal, systemic drop in core body temperature due to prolonged exposure to low environmental temperatures. When heat loss exceeds the body's capacity to generate heat, cellular metabolic rates drop, cardiac output declines, and the central nervous system undergoes severe clinical depression, leading to death.

NODIA

53. (A) Both A and R are true and R is the correct explanation of A.
Prolonged, severe frostbite leads to dry gangrene in peripheral extremities. The pathological basis of this lesion is intense localized vasoconstriction combined with direct cold-induced endothelial cell damage. This vascular disruption triggers platelet adhesion and intravascular thrombosis, completely blocking localized blood flow and causing ischemic coagulative necrosis of the distal skin and tissue structures.

54. (C) Low atmospheric pressure causing systemic hypoxia
In high-altitude environments, the physiological stress is caused by low barometric pressure, which directly reduces the partial pressure of oxygen in the inspired air. This environmental decrease limits oxygen diffusion across the alveolar-capillary membrane, leading to systemic hypoxia, which triggers compensatory physiological responses such as hyperventilation, polycythemia, and pulmonary arterial vasoconstriction.

55. (D) Right-sided heart failure from pulmonary hypertension
Brisket disease, or high-mountain disease in cattle, is a classic manifestation of right-sided heart failure. Chronic hypoxia at high altitudes triggers reflex pulmonary vasoconstriction, leading to pulmonary hypertension. The right ventricle undergoes marked hypertrophy and subsequent dilation to overcome this high vascular resistance, eventually failing and causing severe, dependent subcutaneous edema.

56. (A) Medial hypertrophy of pulmonary arterioles
In chronic altitude sickness, the fundamental vascular alteration is medial hypertrophy of the small pulmonary arterioles. Persistent hypoxia stimulates a marked increase in the smooth muscle thickness within the tunica media of these arterioles. This remodeling narrows the vascular lumen, significantly increasing resistance to pulmonary blood flow and directly driving chronic pulmonary hypertension.

57. (B) Brisket region
High-altitude disease in cattle causes profound, gravity-dependent subcutaneous edema that

is most prominent in the brisket region. This lesion results from right-sided congestive heart failure, which raises systemic venous hydrostatic pressure. Combined with gravity, this increased venous pressure forces fluid out of the microvasculature and into the subcutaneous connective tissue spaces.

58. (B) Lightly pigmented or unpigmented skin
Photosensitization requires three critical factors: a circulating photodynamic compound, exposure to ultraviolet solar radiation, and lightly pigmented or unpigmented skin. Melanin pigment in dark skin protects underlying tissues by absorbing and scattering ultraviolet light. In contrast, unpigmented skin allows these ultraviolet rays to penetrate and excite the photodynamic molecules, causing severe free radical damage.

59. (D) III only
Congenital porphyria is not an infectious viral disease. Instead, it is an inherited metabolic defect in heme synthesis that results in the abnormal accumulation of endogenous photodynamic porphyrins in tissues like the skin, teeth, and bones. Therefore, only the third statement is incorrect, whereas the explanations of primary and secondary photosensitization are correct.

60. (C) Phylloerythrin
Secondary or hepatogenous photosensitization in ruminants is caused by the systemic accumulation of phylloerythrin. Phylloerythrin is a photodynamic metabolite produced during microbial digestion of dietary chlorophyll in the rumen. Normal hepatic function is required to excrete phylloerythrin via bile; however, hepatocellular damage or biliary obstruction leads to its accumulation in blood and unpigmented skin.

61. (D) Chlorophyll
Phylloerythrin is a highly active photodynamic porphyrin compound that is normally formed within the gastrointestinal tract of herbivores. It is produced through the microbial degradation of dietary chlorophyll by rumen micro-organisms. Once absorbed into the portal circulation, it is typically extracted by the liver and excreted in bile under normal physiological conditions.

62. (C) Congenital porphyria
Congenital porphyria is an inherited metabolic disorder characterized by a deficiency in enzymes of the heme biosynthetic pathway, most commonly uroporphyrinogen III cosynthase. This enzymatic block leads to the excessive production and accumulation of photodynamic uroporphyrin and coproporphyrin, which deposit in the skin, bones, and teeth, causing severe, endogenous photosensitization upon light exposure.

63. (B) Pyrimidine (thymine) dimers in DNA
Non-ionizing radiation, particularly ultraviolet solar radiation, lacks the energy to ionize atoms but is absorbed directly by nucleic acids. This absorption induces covalent cross-linking between adjacent pyrimidine bases on a DNA strand, resulting in the formation of cyclobutane pyrimidine dimers, which disrupt DNA replication and transcription, potentially causing cellular mutation or apoptosis.

64. (D) Squamous cell carcinoma
Chronic exposure to solar ultraviolet radiation is a well-established extrinsic predisposing factor for the development of cutaneous squamous cell carcinoma, particularly in poorly pigmented, sparsely haired areas of animals, such as the eyelids of cattle or ears of white cats. UV-induced DNA mutations damage local tumor suppressor genes, driving neoplastic epidermal cell proliferation.

65. (A) P-3, Q-1, R-2, S-4
Physical agents induce distinct pathological lesions in animal tissues. Extreme cold causes ischemic necrosis due to vasoconstriction and microvascular thrombosis. High altitude induces systemic hypoxia and pulmonary hypertension. Ultraviolet light creates pyrimidine dimers within cell nuclei, and extreme heat causes widespread protein denaturation and coagulative necrosis, illustrating how physical forces disrupt normal cellular function.

66. (C) UV radiation
Actinic keratosis is a precancerous epidermal lesion that develops on sun-damaged, lightly pigmented skin due to chronic exposure to solar ultraviolet radiation. The UV light induces progressive mutations in epidermal keratinocytes, causing focal dysplasia, hyperkeratosis, and scaling of the epidermis, which can eventually

Click Here to Buy book on Amazon

progress to invasive squamous cell carcinoma in animals.

67. (A) Radiolysis of cellular water, generating free radicals
Ionizing radiation induces cell injury primarily through the radiolysis of cellular water, a process that generates highly reactive oxygen-derived free radicals and hydroxyl radicals. These reactive species attack and damage critical intracellular macromolecules, particularly double-stranded DNA backbones, cell membrane lipids, and functional enzymes, culminating in genetic mutations, chromosomal aberrations, or mitotic cell death.

68. (D) Dividing cells in bone marrow and intestinal crypts
Cells that are actively dividing and poorly differentiated are highly sensitive to ionizing radiation damage, a concept known as the Law of Bergonie and Tribondeau. Consequently, rapidly proliferating stem cell populations, such as hematopoietic cells in the bone marrow and epithelial cells within intestinal crypts, are the most vulnerable, whereas mature neurons are highly radioresistant.

69. (B) Bone marrow suppression and gastrointestinal sloughing
Acute Radiation Syndrome caused by high-dose whole-body ionizing radiation first manifests as severe bone marrow suppression and gastrointestinal mucosal sloughing. These lesions occur because radiation selectively targets rapidly dividing hematopoietic progenitors and intestinal crypt stem cells. This destruction leads to profound leukopenia, thrombocytopenia, anemia, intractable diarrhea, dehydration, and high susceptibility to fatal systemic septicemia.

70. (C) Internal heating from agitation of water molecules
Microwave radiation is a form of non-ionizing radiation that causes tissue injury primarily by generating internal thermal heat. Microwaves oscillate polarized molecules, primarily water and dissolved ions, causing them to rotate rapidly and collide. This friction-induced molecular agitation generates internal heat, causing localized thermal burns and coagulative tissue necrosis in water-rich organs directly.

NODIA

71. (C) I, II and IV
Alternating current is highly dangerous because it induces continuous tetanic muscle spasms that prevent the animal from releasing the electrical source. Fatalities from electrocution or lightning strikes typically result from acute ventricular fibrillation or respiratory center paralysis. Burns also occur at entry and exit points, whereas lightning does not produce freezing physical injuries.

72. (A) Lichtenberg figures
Lichtenberg figures are transient, branching, fern-like erythematous patterns that can appear on the skin of animals and humans struck by lightning. These markings are not true thermal burns; instead, they result from the passage of high-voltage electrical current through cutaneous vasculature, causing localized capillary dilation and extravasation of red blood cells along the pathway.

73. (B) Electroporation
Electroporation is a non-thermal mechanism of cellular injury caused by exposure to high-voltage electrical currents. The intense electric field alters the electrical potential across cell membranes, creating transient or permanent nanoscale pores within the lipid bilayer. This membrane disruption leads to uncontrolled ion fluxes, loss of cell volume regulation, and subsequent cell death.

74. (B) Lead
Lead toxicity is characterized by encephalopathy and the development of pathognomonic acid-fast intranuclear inclusion bodies in the epithelial cells of proximal renal tubules. These inclusions are complexes of lead and specific nuclear proteins. Identifying these distinct structures histologically in kidney tissue is highly diagnostic, confirming toxic lead exposure in affected animal patients.

75. (A) Copper
Chronic copper toxicosis in sheep is characterized by a sudden hemolytic crisis when stored liver copper is rapidly released into the circulation. This massive copper release causes severe intravascular hemolysis, resulting in hemoglobinemia and hemoglobinuria. The kidneys become dark, swollen, and slate-grey or “gunmetal” colored due to accumulation of hemoglobin within renal tubules.

76. (C) Capillary endothelium, especially in the GI tract
Inorganic arsenic is a potent cellular toxin that primarily targets capillary endothelial cells, particularly within the gastrointestinal tract. By disrupting cell respiration and sulfhydryl-containing enzymes, arsenic causes endothelial necrosis, leading to widespread capillary dilation, increased permeability, congestion, and fluid transudation. This microvascular injury manifests clinically as profuse, hemorrhagic diarrhea, shock, and rapid death.

77. (A) I, II, IV, III
The pathogenesis of chronic copper toxicosis in sheep begins with silent, progressive accumulation of copper in hepatocytes over several months. A sudden stressful event, such as handling or shipping, acts as a trigger, prompting the acute release of stored copper into the bloodstream. This rapid elevation causes massive intravascular hemolysis, hemoglobinuria, and gunmetal kidney development.

78. (C) Central nervous system and renal tubules
Mercury poisoning, in both organic and inorganic forms, primarily targets the central nervous system and the renal tubule epithelium. Mercury binds tightly to sulfhydryl groups on cell enzymes, disrupting cellular respiration and metabolic functions. This biochemical inhibition leads to severe encephalomalacia and neuronal necrosis in the brain, alongside acute tubular necrosis within the kidneys.

79. (B) Fungi
Mycotoxicosis is a form of exogenous organic toxicity caused by the ingestion of toxic secondary metabolites, known as mycotoxins, which are produced by actively growing fungi or molds. These toxigenic fungi, such as Aspergillus, Penicillium, and Fusarium, commonly colonize agricultural crops, grains, and animal feed during production, harvesting, transport, or storage of feed ingredients.

80. (D) Liver
Aflatoxicosis is a severe mycotoxicosis caused by the ingestion of aflatoxins, primarily produced by the fungus Aspergillus flavus. The primary target organ is the liver, where metabolizing enzymes convert aflatoxins into highly reactive, toxic epoxide intermediates. This biochemical activation causes acute hepatocellular necrosis, lipid accumulation, and, under chronic exposure, biliary hyperplasia and hepatic carcinoma.

81. (A) Urinary bladder neoplasms and hematuria
Ingestion of Bracken fern (Pteridium aquilinum) in cattle is classically associated with bone marrow suppression and chronic enzootic hematuria. The plant contains ptaquiloside, a highly toxic carcinogen that induces severe, progressive myelotoxic bone marrow aplasia and directly triggers the development of multiple neoplastic epithelial and mesenchymal tumors in the urinary bladder mucosa, leading to hematuria.

82. (C) Neurotoxins block neuromuscular transmission, while hemotoxins cause coagulopathy and tissue damage.
Snake venoms exhibit distinct pathophysiological mechanisms depending on their chemical composition. Neurotoxic venoms, typically found in elapids, contain polypeptides that block acetylcholine receptors at neuromuscular junctions, causing flaccid paralysis and respiratory failure. Conversely, hemotoxic venoms, found in viperids, contain enzymes that damage vascular endothelium, destroy platelets, and consume clotting factors, causing hemorrhage and necrosis.

83. (D) A is false but R is true.
Ingestion of St. John's wort causes primary, not secondary photosensitization, because the plant contains the preformed photodynamic agent hypericin. However, liver failure prevents the normal clearance of phylloerythrin, an endogenous chlorophyll metabolite, causing it to accumulate in the skin. Thus, the assertion is false but the supporting reason is biologically true.

84. (D) Severe vasoconstriction causing gangrene of extremities
Ergotism, caused by the ingestion of toxic ergot alkaloids produced by the fungus Claviceps purpurea, primarily affects livestock. These alkaloids directly stimulate smooth muscle contraction in arteriolar walls, causing intense, persistent peripheral vasoconstriction. This vascular blockage leads to endothelial damage, intravascular thrombosis, ischemic necrosis, and dry gangrene of the distal extremities.

Downloaded from www.pdf.tube

85. (B) Iatrogenic toxicity
Iatrogenic toxicity refers to any adverse condition, cell injury, or pathological tissue damage in an animal that occurs directly or inadvertently as a result of veterinary medical treatment, surgical intervention, diagnostic procedures, or prescribed therapeutic drug administration. This classification distinguishes veterinarian-induced injuries from naturally occurring genetic, congenital, or infectious disease etiologies.

86. (C) Aminoglycosides
Aminoglycoside antibiotics are highly effective against Gram-negative bacteria but are associated with severe iatrogenic nephrotoxicity and ototoxicity if administered in high doses or over prolonged periods. These drugs accumulate in renal proximal tubular epithelial cells, inducing cellular necrosis, and in the sensory hair cells of the inner ear, causing permanent dysfunction.

87. (D) Inhibiting acetylcholinesterase, causing cholinergic excess
Organophosphates cause severe toxicity by binding covalently to and inhibiting the acetylcholinesterase enzyme at neuromuscular junctions and cholinergic synapses. This inhibition prevents the breakdown of the neurotransmitter acetylcholine, leading to its accumulation and persistent overstimulation of muscarinic and nicotinic receptors, which clinically manifests as excessive secretions, muscle fasciculations, and respiratory failure.

88. (A) Vitamin K
Anticoagulant rodenticides, such as warfarin or brodifacoum, cause fatal internal hemorrhage by competitively inhibiting the enzyme vitamin K epoxide reductase. This inhibition blocks the recycling of active vitamin K, which is essential for the post-translational activation of clotting factors II, VII, IX, and X, leading to severe coagulopathy and extensive internal bleeding.

89. (C) II and IV
Melamine is not an essential amino acid; rather, it is an industrial chemical added illegally to pet foods to artificially inflate crude protein measurements, causing crystal nephropathy. Furthermore, food adulteration includes synthetic chemical contaminants, not just naturally occurring plant toxins. Thus, the second and fourth statements are incorrect, while the other statements are correct.

90. (B) Stomatitis and gastropathy
High systemic levels of uremic toxins, such as blood urea nitrogen and creatinine, lead to the clinical syndrome of uremia. In uremic animals, oral micro-organisms degrade blood urea into irritating ammonia, causing acute ulcerative stomatitis. Additionally, uremia induces microvascular endothelial injury, resulting in ischemic, ulcerative, and hemorrhagic gastropathy across the stomach.

91. (A) Diabetes mellitus or negative energy balance
Ketoacidosis is a severe metabolic condition that develops when cells are deprived of glucose, typically due to diabetes mellitus or extreme negative energy balance. Lacking intracellular glucose, the body undergoes intensive lipolysis, oxidizing free fatty acids into excess ketone bodies like acetoacetate and beta-hydroxybutyrate, which overwhelm blood buffering capacities and lower systemic pH.

92. (C) Icterus (Jaundice)
Icterus, or jaundice, is a clinical and pathological condition characterized by yellow discoloration of the skin, sclera, mucous membranes, and internal organs. This staining is caused by the systemic accumulation of bilirubin in the blood and tissue fluids, occurring when bilirubin production exceeds hepatic clearance, conjugation, or excretion physiological pathways.

93. (D) Pre-hepatic (hemolytic) icterus
Pre-hepatic, or hemolytic, icterus develops when accelerated destruction of erythrocytes releases massive amounts of hemoglobin into the bloodstream. This rapid hemoglobin breakdown generates unconjugated bilirubin at a rate that completely overwhelms the liver's physiological capacity to conjugate and excrete it, leading to systemic accumulation of unconjugated bilirubin and tissue discoloration.

NODIA

94. (C) Bile duct obstruction preventing bilirubin excretion
Post-hepatic, or obstructive, icterus is caused by physical blockages in the biliary system that prevent the normal excretion of conjugated bilirubin into the intestine. Blockages, such as gallstones, neoplasia, or biliary parasites, force conjugated bilirubin to reflux back into hepatic sinusoids and enter systemic circulation, raising blood levels and tissue staining.

95. (B) P-2, Q-4, R-1, S-3
Icterus is classified by its specific physiological mechanism. Pre-hepatic icterus is caused by massive erythrocyte destruction, hepatic icterus is driven by hepatocellular damage that impairs conjugation, post-hepatic icterus results from physical biliary tract obstruction, and neonatal isoerythrolysis represents maternal antibody-mediated hemolysis in newborn animals, causing acute pre-hepatic hemolytic icterus within affected foals.

96. (D) Antibodies binding directly to cell surface antigens
Type II hypersensitivity reactions, also known as antibody-dependent or cytotoxic hypersensitivity, occur when IgG or IgM antibodies bind directly to specific antigens present on the surface of host cells or tissues. This direct antibody binding initiates cell destruction by activating the complement cascade, promoting phagocytosis via opsonization, or recruiting cytotoxic tissue effector cells directly.

97. (A) Complement Membrane Attack Complex causing cell lysis
In Type II hypersensitivity, target cell destruction is commonly mediated by the activation of the classical complement pathway. When IgM or IgG binds to cell-surface antigens, it triggers a cascade that culminates in the assembly of the Membrane Attack Complex on the cell membrane, creating trans-membrane channels that cause uncontrolled osmotic cell lysis.

98. (C) Leukocytes destroying target cells coated with specific antibodies
Antibody-dependent cellular cytotoxicity is a major immunological mechanism of target cell destruction. In this process, host cells are first coated with specific IgG antibodies. Non-specific cytotoxic leukocytes, such as Natural Killer cells, macrophages, and neutrophils, bind to the Fc portions of these bound antibodies, triggering the release of lytic enzymes to lyse the target cell.

99. (A) Autoimmune hemolytic anemia
Autoimmune hemolytic anemia is a classic clinical example of a cytolytic Type II hypersensitivity reaction in animals. In this disease, the host produces autoantibodies against its own erythrocyte membrane antigens, leading to premature red blood cell clearance by splenic macrophages via phagocytosis, or direct intravascular hemolysis through complement activation pathways.

100. (D) Type II hypersensitivity
Neonatal isoerythrolysis in newborn foals is a direct manifestation of Type II cytotoxic hypersensitivity. It occurs when a foal ingests colostrum containing maternal antibodies directed specifically against the foal's inherited paternal red blood cell antigens. Once absorbed, these antibodies bind to the foal's erythrocytes, initiating complement activation, massive intravascular hemolysis, and hemolytic icterus.

101. (A) II, IV, I, III
The sequence of complement-mediated Type II hypersensitivity begins with the binding of IgG or IgM to a specific cell surface antigen. This antigen-antibody interaction triggers the activation of the classical complement cascade. Complement activation culminates in the assembly of the Membrane Attack Complex on the lipid bilayer, which initiates fatal osmotic target cell lysis.

102. (C) Deposition of circulating soluble immune complexes in tissues
Type III hypersensitivity is mediated fundamentally by the formation of soluble antigen-antibody (immune) complexes, typically in slight antigen excess, which circulate and deposit within tissue microvasculature. Once deposited, these immune complexes activate the classical complement pathway, recruiting polymorphonuclear neutrophils that release lysosomal enzymes and reactive oxygen species, inducing severe, localized necrotizing tissue vasculitis.

103. (D) Small blood vessel walls, renal glomeruli, and synovial joints
In Type III hypersensitivity, circulating soluble immune complexes are mechanically filtered and deposit primarily within the walls of small blood vessels, renal glomerular basement membranes, and synovial joint membranes. These specialized filtration structures are highly susceptible to deposition, which triggers localized complement activation, neutrophil influx, and subsequent necrozing inflammatory tissue damage processes.

Click Here to Buy book on Flipkart

104. (A) Arthus reaction
The Arthus reaction is a classic, localized form of Type III hypersensitivity. It is induced by injecting an antigen intradermally into an animal that has high levels of circulating antibodies. The local antigen-antibody complexes precipitate in the walls of dermal blood vessels, activating complement, recruiting neutrophils, and causing localized vasculitis, edema, and tissue necrosis.

105. (B) Systemic Type III reaction
Serum sickness is a classic systemic Type III hypersensitivity reaction. It occurs when a large dose of foreign protein or serum is administered. As the host produces antibodies, soluble immune complexes form in slight antigen excess, circulate systemically, and deposit in multiple tissue beds, causing generalized vasculitis, glomerulonephritis, and arthritis several days later.

106. (C) Lysosomal enzymes and reactive oxygen species from recruited neutrophils
Type III hypersensitivity is initiated by the deposition of antigen-antibody complexes in tissues. This deposition triggers the activation of the complement system, which recruits neutrophilic granulocytes to the local area. The severe injury to local structures is directly executed by the subsequent release of active lysosomal enzymes and destructive oxygen-derived free radicals from these activated inflammatory cells.

107. (D) II and III
Type III hypersensitivity is mediated by IgG or IgM antibodies rather than IgE. The deposition of circulating antigen-antibody complexes in vessel walls activates the complement cascade. This activation recruits neutrophils, causing severe inflammatory damage and vasculitis characterized by fibrinoid necrosis. Therefore, complement activation and fibrinoid necrosis of blood vessels represent critical, defining components of this specific pathological pathway.

108. (C) Type III hypersensitivity
Fibrinoid necrosis of blood vessels represents a distinctive form of vascular damage typical of immune complex-mediated diseases. This pathological lesion occurs when antigen-antibody complexes deposit within the walls of arteries and veins, initiating complement activation and leukocyte recruitment. The deposition of plasma proteins, including fibrin, alongside immunoglobulins, results in a bright pink, amorphous appearance on routine histologic evaluation.

109. (B) Any chemical species with an unpaired electron in its outer orbit
A free radical is defined as any highly reactive chemical species that possesses one or more unpaired electrons in its outer atomic orbit. The presence of this unpaired electron makes the molecule extremely unstable and prone to initiating chain reactions. These species rapidly interact with adjacent organic or inorganic molecules to acquire stability, thereby inducing significant cellular damage.

110. (B) Mitochondrial oxidative phosphorylation
Reactive oxygen species are generated as normal physiological byproducts during mitochondrial oxidative phosphorylation. Under physiological conditions, the transfer of electrons through the electron transport chain is slightly imperfect, leading to the univalent reduction of molecular oxygen. This process continuously produces small amounts of superoxide anions, which are subsequently processed by endogenous cellular antioxidant defense mechanisms.

111. (A) Superoxide anion ($O_2^{\cdot -}$)
The superoxide anion is a highly reactive free radical generated through the univalent reduction of molecular oxygen. This physiological event occurs primarily within the mitochondria during normal cellular respiration or via enzymatic activity in activated phagocytes. It serves as the primary precursor for many other reactive oxygen species and initiates cellular oxidative stress when antioxidant defenses are overwhelmed.

112. (C) Fenton reaction
Hydrogen peroxide is a relatively stable, non-radical reactive oxygen species that readily diffuses across cellular membranes. However, its intracellular pathogenicity lies in its capacity to undergo transition metal-catalyzed chemical reduction. In the presence of reduced iron, it undergoes a specific chemical reaction to generate the highly reactive and destructive hydroxyl radical, which causes severe cellular damage.

113. (D) Both A and R are false.
Both statements regarding hydrogen peroxide are false. It is not biologically harmless, as it can

Downloaded from www.pdf.tube

be converted into the extremely toxic hydroxyl radical, which causes substantial cellular damage. Furthermore, it is not a direct free radical itself, nor does it immediately destroy cellular membranes without undergoing transition metal-catalyzed reactions like the Fenton process to generate radicals.

114. (C) Iron (Fe^{2+})
The Fenton reaction is a critical chemical process that converts hydrogen peroxide into the highly toxic and reactive hydroxyl radical. This transition metal-catalyzed reaction relies heavily on the presence of reduced iron acting as an electron donor. Intracellular availability of free iron therefore directly influences the rate of hydroxyl radical generation and subsequent oxidative damage within cellular systems.

115. (B) Direct inhibition of ATP synthase via competitive enzyme binding
Free radicals induce cellular injury through lipid peroxidation of membranes, oxidative modification of proteins, and severe DNA damage. They do not act as direct, competitive inhibitors of the ATP synthase enzyme. Instead, mitochondrial dysfunction and decreased ATP production resulting from free radical exposure are consequences of structural membrane damage and overall organelle degradation rather than competitive binding.

116. (D) Membrane polyunsaturated fatty acids
Lipid peroxidation is a major mechanism of cell injury initiated by reactive oxygen species. Free radicals aggressively attack and abstract hydrogen atoms from the double bonds of polyunsaturated fatty acids located within cellular and organellar membranes. This interaction generates unstable lipid radicals, initiating a self-propagating chain reaction that destroys membrane integrity, disrupts ion gradients, and leads to cell death.

117. (A) Malondialdehyde (MDA)
Lipid peroxidation of cellular membranes produces highly reactive and cytotoxic aldehydes as breakdown products. Malondialdehyde is a prominent, stable end-product of this destructive pathway and serves as a classic, reliable biomarker for assessing oxidative stress in tissues. Measuring its concentration allows researchers and pathologists to quantify the extent of free radical-mediated damage to membrane lipids.

118. (C) By promoting sulfhydryl-mediated cross-linking and fragmentation
Reactive oxygen species induce severe damage to cellular proteins primarily by promoting the oxidation of amino acid side chains. This oxidative modification leads to the formation of protein-protein cross-links, particularly through sulfhydryl group oxidation, as well as polypeptide fragmentation. Consequently, these structurally altered proteins undergo rapid enzymatic degradation or lose their normal structural and functional enzymatic capabilities.

119. (A) II and IV
Oxidative DNA damage is highly relevant to cellular aging, genomic instability, and neoplastic transformation because mutations accumulate over time. Reactive oxygen species, particularly hydroxyl radicals, weaken and break nucleotide bonds, rather than strengthening them. Thus, statements claiming that oxidative damage is irrelevant to aging or that it enhances genomic stability by strengthening bonds are completely incorrect.

120. (D) Enzymatic inactivation and degradation of vital structural proteins
Extensive protein cross-linking induced by reactive oxygen species causes severe structural modifications in essential cellular proteins. This oxidative damage leads to the inactivation of vital enzymes, disruption of structural proteins, and misfolding of critical polypeptides. Cells respond by targeting these damaged, non-functional proteins for rapid enzymatic degradation, culminating in a significant loss of normal physiological cell functions.

121. (B) Superoxide dismutase (SOD)
Superoxide dismutase is a vital enzymatic antioxidant that serves as the first line of defense against reactive oxygen species. This enzyme specifically catalyzes the dismutation of the highly reactive superoxide anion into oxygen and hydrogen peroxide. This critical conversion prevents superoxide from participating in other damaging chemical reactions, preparing it for detoxification by other cellular enzymes.

122. (C) Catalase
Catalase is a major endogenous antioxidant enzyme highly concentrated within cellular peroxisomes. It plays a critical role in cellular detoxification by directly converting hydrogen

NODIA

peroxide into harmless water and molecular oxygen. This enzymatic action prevents the accumulation of hydrogen peroxide, thereby blocking its conversion into the highly destructive hydroxyl radical via transition metal-catalyzed reactions.

123. (D) Selenium
Glutathione peroxidase is a vital intracellular antioxidant enzyme that detoxifies lipid hydroperoxides and hydrogen peroxide. To function efficiently, this enzyme requires the essential trace mineral selenium as a critical cofactor, which is incorporated as selenocysteine at the active site. A deficiency in selenium severely compromises cellular defense mechanisms, leading to increased susceptibility to oxidative damage.

124. (A) Vitamin E (alpha-tocopherol)
Vitamin E is a major, lipid-soluble, non-enzymatic antioxidant concentrated within cellular and organellar membranes. It protects these lipid bilayers from oxidative damage by directly scavenging lipid peroxyl radicals, thereby functioning as a highly effective chain breaker. By terminating the self-propagating cycle of lipid peroxidation, it preserves membrane structural integrity and prevents premature cell death.

125. (B) P-2, Q-4, R-1, S-3
The major cellular antioxidants occupy distinct physiological locations to neutralize reactive oxygen species. Superoxide dismutase converts superoxide in the cytosol and mitochondria, while catalase decomposes hydrogen peroxide within peroxisomes. Vitamin E acts as a key lipid-soluble scavenger directly inside membranes, whereas Vitamin C functions in the aqueous phase to neutralize free radicals and regenerate other antioxidants.

126. (B) Scavenging aqueous free radicals and regenerating oxidized Vitamin E
Vitamin C is a vital water-soluble antioxidant that operates primarily in the aqueous compartments of cells and extracellular fluids. It directly scavenges free radicals, preventing them from damaging cellular components. Additionally, it plays a key role in regenerating the active, reduced form of Vitamin E from its oxidized state, thereby maintaining lipid membrane defenses against peroxidation.

127. (A) Nutritional muscular dystrophy (White muscle disease)
A severe dietary deficiency of Vitamin E or selenium in livestock compromises cellular antioxidant defense systems. This vulnerability allows uncontrolled lipid peroxidation of cell membranes, particularly in highly active tissues like skeletal and cardiac muscle. The resulting oxidative damage leads to myofiber necrosis, presenting clinically and pathologically as nutritional muscular dystrophy, commonly known as white muscle disease.

128. (C) Peroxynitrite ($ONOO^-$)
Nitric oxide is a soluble gas that functions as a cellular signaling molecule. However, under inflammatory conditions, it can react at an extremely rapid, diffusion-controlled rate with the superoxide anion. This chemical combination generates peroxynitrite, a highly reactive nitrogen species that directly damages lipids, proteins, and DNA, thereby contributing significantly to inflammatory tissue injury.

129. (D) Reactive oxygen species generation overwhelming cellular antioxidant defenses
Oxidative stress is defined as a cellular state wherein the generation of reactive oxygen species exceeds the capacity of endogenous antioxidant defense systems. This imbalance results in the accumulation of free radicals, leading to oxidative damage of lipids, proteins, and DNA. It serves as a key pathogenic mechanism in numerous degenerative, inflammatory, and neoplastic diseases.

130. (C) It has high oxygen use and membranes rich in polyunsaturated lipids.
The central nervous system is exceptionally vulnerable to oxidative stress due to its unique physiological characteristics. It consumes a disproportionately large amount of oxygen relative to its mass, generating high levels of mitochondrial byproducts. Furthermore, neuronal membranes are highly enriched with polyunsaturated fatty acids, which serve as prime substrates for devastating lipid peroxidation reactions.

131. (B) II, III and IV
Intracellular antioxidant defenses rely on specific spatial and chemical properties. Glutathione peroxidase requires selenium to function, Vitamin E protects lipid bilayers from peroxidation, and

Click Here to Buy book on Amazon

superoxide dismutase converts superoxide to hydrogen peroxide. Catalase is concentrated in peroxisomes rather than lysosomes, and Vitamin C functions as an antioxidant scavenger rather than promoting hydroxyl radical generation.

132. (D) Fixation
Fixation is the foundational process in pathology used to preserve tissues immediately after death or removal from the body. It halts enzymatic autolysis and prevents bacterial putrefaction by cross-linking proteins and stabilizing cellular structures. This preservation maintains the architectural and cytological morphology of the tissue, allowing for accurate microscopic evaluation and diagnostic analysis.

133. (A) Nuclear dissolution (Karyolysis)
Karyolysis represents the enzymatic dissolution of nuclear chromatin and is a definitive, microscopic hallmark of absolute, irreversible cell death. Unlike reversible changes such as cellular swelling or chromatin clumping, the complete loss of the nucleus indicates that the cell has progressed past the point of recovery and has undergone necrosis, leading to permanent loss of function.

134. (C) Cellular pathology
Cellular pathology is a foundational scientific concept establishing that all diseases originate at the cellular level. This principle asserts that macroscopic clinical signs and structural tissue alterations observed during disease are direct consequences of functional and morphological abnormalities within individual cells. It shifted medical understanding away from humoral imbalances to localized cellular dysfunction.

135. (A) Molecular pathology
Molecular pathology is a specialized branch of diagnostic medicine focused on identifying diseases by analyzing nucleic acids and proteins. It utilizes advanced techniques, such as polymerase chain reaction and immunohistochemistry, to detect genetic mutations, viral DNA, or altered protein expression. This discipline complements traditional microscopic evaluation, providing highly precise diagnostic, prognostic, and therapeutic information.

136. (D) The idiopathic disease has an unknown etiology, whereas the acquired disease has a defined external cause.
The fundamental distinction between idiopathic and acquired diseases lies in their etiology. An idiopathic disease is characterized by an unknown or unidentifiable cause, arising spontaneously without a clear origin. Conversely, an acquired disease develops after birth due to a defined external factor, such as a viral infection, chemical exposure, physical trauma, or nutritional deficiency.

137. (A) Both A and R are true and R is the correct explanation of A.
Starvation acts as a profound extrinsic predisposing factor for infectious diseases. Severe nutritional deprivation compromises the host immune system by reducing the production of essential protective proteins, antibodies, and functional leukocytes. This immunosuppression directly diminishes resistance to animate pathogens, making the starved animal highly susceptible to developing severe, life-threatening infections from opportunistic organisms.

NODIA

138. (C) Compartment syndrome / Crush syndrome
Prolonged physical compression or crushing of skeletal muscle tissue causes severe ischemic and mechanical damage to myofibers. This injury results in the rapid leakage of intracellular contents, particularly myoglobin and potassium, into the systemic circulation. This pathological state is clinically and pathologically defined as compartment syndrome or crush syndrome, which can lead to life-threatening systemic complications.

139. (D) Acute renal failure due to myoglobinuric nephrosis
Massive release of myoglobin into the bloodstream following severe skeletal muscle trauma leads to myoglobinuria. Once filtered by the glomeruli, high concentrations of myoglobin cause direct toxic damage to renal tubular epithelial cells and form obstructing casts within the nephrons. This sequence of events results in acute renal failure, pathologically classified as myoglobinuric nephrosis.

140. (A) Puncture
A puncture is a physical wound characterized by a depth that is significantly greater than its surface length, typically caused by a narrow,

pointed object. This type of injury can carry pathogens or foreign material deep into tissues, creating an anaerobic environment that favors the growth of specific bacteria, while showing minimal superficial damage.

141. (B) Cardiovascular collapse from vasodilation and hemoconcentration
Acute heat stroke causes severe systemic hyperthermia, which triggers widespread peripheral vasodilation as the body attempts to dissipate heat. This marked vascular expansion, combined with rapid fluid loss and subsequent hemoconcentration, severely compromises venous return. The immediate cause of death is cardiovascular collapse, as the heart fails to maintain adequate perfusion to vital organs.

142. (B) Prolonged vasoconstriction leading to edema, endothelial damage, and blistering
Trench foot is a severe, non-freezing cold injury resulting from prolonged exposure to wet, cold conditions. The pathological hallmark involves sustained, intense vasoconstriction of the microvasculature. This prolonged lack of blood flow leads to severe hypoxia, endothelial cell damage, localized edema, and blistering of the skin, without the physical crystallization of intracellular water.

143. (A) High ambient humidity and strong winds
Exposure to low ambient temperatures can rapidly lead to clinical hypothermia if heat loss exceeds the body's heat-generating capacity. Environmental factors such as high humidity and strong winds vastly accelerate this process by enhancing convective and evaporative heat loss. These conditions quickly strip away the protective boundary layer of warm air surrounding the animal's body.

144. (C) Reduced availability of oxygen to active cells and tissues
Hypoxia is defined as a pathological state characterized by the reduced availability or delivery of oxygen to active cells and tissues. Unlike anoxia, which denotes a complete absence of oxygen, hypoxia represents a subnormal level that is insufficient to sustain normal aerobic mitochondrial respiration, thereby forcing cells to transition to anaerobic metabolic pathways.

145. (D) Increasing the production of oxygen-carrying red blood cells
To compensate for chronic hypoxia at high altitudes, the body senses the reduced oxygen tension and stimulates the renal secretion of erythropoietin. This hormone acts directly on the bone marrow to accelerate erythropoiesis, significantly increasing the production of oxygen-carrying red blood cells. This polycythemia enhances the blood's oxygen-carrying capacity to maintain systemic tissue perfusion.

146. (C) Reactive oxygen species and free radicals
A photodynamic agent is a chemical compound that accumulates in the skin and absorbs specific wavelengths of light. Upon activation by ultraviolet radiation, the molecule transfers energy to oxygen, directly generating highly damaging reactive oxygen species and free radicals. These reactive molecules initiate lipid peroxidation of local cell membranes, resulting in severe cutaneous inflammation and necrosis.

147. (B) Lack of circumocular pigmentation
Squamous cell carcinoma of the eye and surrounding structures in cattle is highly influenced by breed genetics. A lack of circumocular pigmentation serves as a major intrinsic predisposing factor for this neoplasm. Unpigmented skin lacks the protective barrier of melanin, allowing ultraviolet radiation to directly penetrate epidermal cells and induce mutagenic DNA damage, initiating carcinogenesis.

148. (D) Epidermal melanin synthesis and distribution by melanocytes
The synthesis and distribution of melanin by epidermal melanocytes represents the primary defense mechanism against non-ionizing ultraviolet radiation. Melanin acts as a physical shield over the nuclei of keratinocytes, absorbing and dissipating harmful radiation. This protective pigment prevents ultraviolet rays from directly interacting with genomic DNA and inducing mutagenic pyrimidine dimers, protecting the skin.

149. (A) Possess high energy and penetrance, ionizing deep intracellular molecules
Gamma rays possess extremely high energy and deep tissue penetrance compared to ultraviolet light, which is largely absorbed superficially. This high penetrance allows gamma radiation to reach deep internal organs, where it directly ionizes

Downloaded from www.pdf.tube

water and other intracellular molecules. This ionization produces a cascade of highly reactive free radicals that damage DNA, proteins, and critical subcellular structures.

150. (C) Development of secondary malignancies due to permanent DNA mutations
Ionizing radiation induces DNA damage both through direct interactions and by generating reactive free radicals. If the cell survives this initial insult, unrepaired or misrepaired double-strand breaks can lead to permanent genomic instability. Over time, these somatic mutations accumulate in surviving cell lineages, presenting years later as a high risk for the development of secondary malignancies.

151. (A) The voltage, current type, tissue resistance, and pathway of the current
The severity of tissue injury sustained from an electric shock is determined by several physical and biological parameters. The voltage, current type, tissue resistance, and pathway of the current through the body dictate the extent of internal damage. These factors govern how much electrical energy is converted into thermal energy, causing deep tissue coagulation and disruption.

152. (D) Blood and nerve tissues
During electrocution, the electrical current preferentially follows the path of least physical resistance through the body. Blood and nerve tissues possess high water and electrolyte content, giving them the lowest electrical resistance among body tissues. Consequently, they conduct current easily, suffering immediate and severe functional disruption, including cardiac arrhythmias and neurological failure.

153. (B) Inorganic agents lack carbon-hydrogen bonds, whereas organic agents are complex carbon-based compounds.
The fundamental difference between exogenous inorganic and organic chemical agents lies in their basic chemical structure. Inorganic agents, such as heavy metals and acids, lack carbon-hydrogen bonds and possess simpler elemental structures. In contrast, organic chemical agents are complex carbon-based compounds, including synthetic pesticides, plant toxins, and industrial solvents, which interact differently with biological systems.

154. (C) Basophilic stippling of erythrocytes and intranuclear inclusions in renal tubules
Chronic lead toxicity is characterized by distinct morphological alterations within the hematopoietic and renal systems. Lead directly inhibits enzymes involved in heme synthesis, causing the accumulation of ribosomal RNA aggregates in erythrocytes, visualized as basophilic stippling. Additionally, lead-protein complexes form diagnostic, acid-fast, intranuclear inclusion bodies within the epithelial cells of the renal tubules.

155. (D) The systemic microvasculature, causing hemorrhage and edema
Arsenic toxicity is highly destructive because it targets cellular respiration and capillary integrity. It binds sulfhydryl groups, disrupting critical metabolic enzymes and severely damaging the systemic microvasculature. This vascular injury leads to widespread endothelial necrosis, resulting in massive capillary leakage, extensive hemorrhage, and severe edema, particularly within the gastrointestinal tract and parenchymal organs.

156. (A) Glucocorticoids (steroids)
Iatrogenic hyperadrenocorticism is a metabolic disorder induced by medical intervention. It most commonly results from the prolonged or excessive administration of exogenous glucocorticoids to treat chronic inflammatory or autoimmune conditions. This external hormone supply suppresses the hypothalamic-pituitary-adrenal axis, leading to bilateral adrenal cortical atrophy alongside clinical signs of cortisol excess.

157. (B) Melamine and cyanuric acid
Melamine and cyanuric acid are industrial compounds that, when combined in pet food, act as severe nephrotoxins. Once ingested, they are absorbed and filtered by the kidneys, where they react in the distal tubules to form insoluble, green-brown, lattice-like crystals. These crystals cause mechanical blockage and acute tubular necrosis, leading to rapid, catastrophic renal failure.

NQDIA

158. (B) Hepatoencephalopathy
Severe hepatic failure compromises the liver's ability to detoxify metabolic byproducts, leading to the accumulation of toxic ammonia in the systemic circulation. Ammonia readily crosses the blood-brain barrier, disrupting astrocyte

function, neurotransmission, and osmotic balance within the central nervous system. This metabolic derangement causes a severe, life-threatening neurological syndrome known as hepatoencephalopathy.

159. (A) Salivary and gastric bacteria convert urea into caustic ammonia, severely burning the mucosa.
In animals with severe uremia, high concentrations of urea diffuse into the saliva and gastrointestinal secretions. Resident bacteria containing the enzyme urease quickly hydrolyze this urea into caustic ammonia. This localized production of ammonia is highly irritating and directly burns the mucosal lining, resulting in painful oral ulcerations, stomatitis, and hemorrhagic gastritis.

160. (C) Denaturing systemic proteins and impairing enzymatic activity
Ketoacidosis is a severe metabolic state characterized by the accumulation of acidic ketone bodies, which lowers systemic blood pH. This metabolic acidosis disrupts cellular homeostasis by altering the electrical charge on proteins. The resulting denaturation of structural and enzymatic proteins impairs critical metabolic pathways and cellular functions, leading to systemic depression and organ dysfunction.

161. (D) A complete mechanical blockage of the common bile duct
Post-hepatic icterus, or obstructive jaundice, occurs when there is an interruption in the normal flow of conjugated bilirubin from the liver to the duodenum. This is most frequently caused by a complete mechanical blockage of the common bile duct by gallstones, tumors, or parasites, leading to systemic accumulation of bile pigments.

162. (B) Type II hypersensitivity
Blood transfusion reactions resulting from the administration of mismatched blood are classic examples of Type II hypersensitivity. Pre-existing host antibodies directly bind to foreign antigens present on the donor red blood cells. This antigen-antibody interaction triggers complement activation and opsonization, leading to rapid intravascular hemolysis or extravascular phagocytosis of the transfused cells.

163. (B) Target cells are antibody-coated to enhance phagocytosis
In Type II hypersensitivity, opsonization is a critical immunological process where target cells are coated with specific antibodies or complement fragments. This coating serves as a powerful signal that enhances the recognition and binding of the target cell by phagocytic leukocytes, such as macrophages, facilitating its rapid engulfment and subsequent intracellular destruction.

164. (D) IgG and IgM
The primary antibody classes involved in initiating Type II and Type III hypersensitivity reactions are IgG and IgM. These immunoglobulins possess specific constant regions that, upon binding to their respective antigens, are capable of binding and activating the C1 component of the complement cascade. This activation initiates the classical complement pathway, promoting inflammation and cell lysis.

165. (A) Severe acute necrotizing vasculitis with local hemorrhage and thrombosis
The Arthus reaction is a classic localized model of Type III hypersensitivity. It is characterized pathologically by severe, acute necrotizing vasculitis, which occurs when antigen-antibody complexes deposit directly within local vessel walls. This deposition triggers intense complement activation, leading to neutrophil recruitment, subsequent vessel wall destruction, localized hemorrhage, and microvascular thrombosis.

166. (C) Activate complement to recruit neutrophils that release destructive proteases and ROS
Immune complexes that deposit within the renal glomeruli initiate severe tissue damage by activating the complement cascade. This activation produces potent chemoattractants, which recruit neutrophils to the glomerular capillaries. These recruited inflammatory cells release highly destructive lysosomal proteases and reactive oxygen species, causing extensive damage to the glomerular basement membrane and podocytes.

167. (B) The abstraction of a hydrogen atom from a polyunsaturated fatty acid by a reactive free radical
The initiation phase of lipid peroxidation begins when a highly reactive free radical abstracts a hydrogen atom from a methylene carbon of a polyunsaturated fatty acid. This abstraction

Click Here to Buy book on Flipkart

leaves behind an unpaired electron on the carbon atom, converting the lipid into an unstable lipid radical, which then undergoes structural rearrangement to form a conjugated diene.

168. (D) Lipid peroxidation propagation
During lipid peroxidation, the propagation phase represents a self-sustaining chain reaction. It occurs when carbon-centered lipid radicals react with molecular oxygen to form lipid peroxyl radicals. These peroxyl radicals then abstract hydrogen atoms from adjacent polyunsaturated fatty acids, generating lipid hydroperoxides and new lipid radicals, continuously spreading damage across the membrane.

169. (B) Hypochlorous acid (HOCl)
During the active respiratory burst in activated neutrophils, the enzyme myeloperoxidase is released into phagolysosomes. This enzyme utilizes hydrogen peroxide and halide ions, particularly chloride, to catalyze the production of hypochlorous acid. Hypochlorous acid is an extremely potent, bactericidal oxidant that rapidly destroys bacterial cell walls and proteins, playing a major role in microbicidal defense.

170. (C) Phospholipid bilayers of cell and organelle membranes
Lipid peroxidation specifically targets the polyunsaturated fatty acids that constitute the structural backbone of biological membranes. Consequently, the phospholipid bilayers of the plasma membrane, mitochondria, and endoplasmic reticulum are the most immediate cellular structures structurally compromised by this oxidative process. This damage leads to a loss of membrane permeability and cell lysis.

171. (D) Superoxide and hydrogen peroxide
The Haber-Weiss reaction is an important chemical process that generates the highly reactive hydroxyl radical in biological systems. This reaction occurs when the superoxide anion directly reacts with hydrogen peroxide. The process is often accelerated in vivo by the presence of trace transition metals like iron, which cycle between oxidized and reduced states to facilitate electron transfer.

172. (A) Undergoes programmed cell death (apoptosis) or necrosis
When intracellular levels of reactive oxygen species overwhelm the cell's antioxidant defenses, severe and irreversible oxidative damage to lipids, proteins, and DNA occurs. Under these conditions, the cell cannot maintain metabolic homeostasis or structural integrity. It typically activates programmed cell death pathways, undergoing apoptosis, or succumbs to passive, inflammatory cell death via necrosis.

173. (B) To safely donate electrons to free radicals, neutralizing them
The fundamental role of the endogenous cellular antioxidant system is to maintain redox homeostasis by neutralizing free radicals before they damage vital macromolecules. Antioxidants achieve this by safely donating electrons to unstable, reactive species, thereby stabilizing them. This protective donation halts damaging chain reactions, such as lipid peroxidation, without rendering the antioxidant itself unstable.

174. (B) Transferferrin
Free, unbound iron is highly dangerous in biological systems because it catalyzes the formation of toxic hydroxyl radicals via the Fenton reaction. Transferrin is a crucial plasma glycoprotein that serves as an antioxidant defense by binding and transporting circulating ferric iron. By keeping iron safely bound, it prevents participation in radical-generating chemical reactions.

NODIA

175. (B) Glutathione reductase
Glutathione reductase is a vital antioxidant enzyme responsible for maintaining high intracellular levels of reduced glutathione. This enzyme catalyzes the reduction of oxidized glutathione disulfide back to its active, reduced sulfhydryl form. To perform this reaction, it utilizes reducing equivalents provided by NADPH, which is primarily generated through the pentose phosphate pathway.

176. (B) Reduced glutathione (GSH)
Glutathione peroxidase protects cells from oxidative damage by reducing hydrogen peroxide and lipid hydroperoxides to water and harmless alcohols. To achieve this, the enzyme oxidizes its essential tripeptide cofactor, reduced glutathione, into glutathione disulfide. This reaction represents a vital defense mechanism for maintaining membrane integrity and preventing the initiation of lipid peroxidation.

177. (D) Normal, healthy mitosis
Prolonged and uncontrolled generation of reactive oxygen species causes extensive damage to cellular components, contributing to the pathogenesis of aging, ischemia-reperfusion injury, and carcinogenesis. However, reactive oxygen species are not implicated in normal, healthy mitosis, which is a tightly regulated physiological process of cell division that relies on balanced signaling pathways and intact genomic structures.

178. (C) Failure of the Na^+/K^+ ATPase pump, causing sodium and water influx
Cellular swelling is the earliest morphologically detectable change in reversible cell injury. It is primarily caused by an acute impairment of mitochondrial ATP production. This energy depletion leads to the failure of the active sodium-potassium ATPase pump, resulting in the intracellular accumulation of sodium ions and a corresponding osmotic influx of water into the cytoplasm.

179. (B) Karyorrhexis
Karyorrhexis is a classic morphological change characterizing irreversible cell death and necrosis. During this process, the chromatin of the dying cell's nucleus condenses and subsequently undergoes enzymatic fragmentation, breaking apart into numerous small, dense, basophilic particles. This nuclear fragmentation indicates complete loss of genomic integrity and signifies that the cell has progressed to necrosis.

180. (D) Pyknosis
Pyknosis is a prominent morphological feature of irreversible cell injury and necrosis. It is characterized by the progressive shrinkage of the nucleus and the extreme condensation of its chromatin into a small, dark, structureless, basophilic mass. This nuclear condensation is often followed by fragmentation or dissolution, representing a clear histological indicator of cell death.

181. (A) Preservation of cell outlines and tissue architecture despite cell death
Coagulative necrosis is a form of cell death typically caused by sudden ischemia or thermal injury. Under the microscope, it is characterized by the temporary preservation of the basic cell outlines and overall tissue architecture. This occurs because the injury denatures both structural proteins and enzymatic proteins, preventing the immediate autolysis and dissolution of the dead cells.

182. (C) Liquefactive necrosis
Liquefactive necrosis is characterized by the rapid and complete enzymatic digestion of dead tissue into a viscous, liquid mass. This process is highly typical of hypoxic injury in the central nervous system, as well as localized bacterial or fungal infections. The abundant release of hydrolytic enzymes from inflammatory cells or autolytic processes completely destroys all structural architecture.

183. (A) Dry, friable, and "cheese-like"
Caseous necrosis is a distinct form of cell death most commonly associated with chronic granulomatous infections, such as tuberculosis. Grossly, the necrotic area is characterized by a dry, friable, yellowish-white appearance that resembles crumbly cheese. Microscopically, the tissue architecture is completely obliterated, leaving behind a granular, amorphous, eosinophilic mass of cellular debris.

184. (D) Dystrophic calcification
Dystrophic calcification refers to the deposition of calcium salts in dead, dying, or degenerating tissues. Crucially, this pathological process occurs in the absence of systemic calcium imbalances, meaning that serum calcium levels remain completely normal. The mineral deposition is initiated by the accumulation of calcium in damaged mitochondria and its binding to membrane phospholipids.

NODIA

185. (B) Persistent hypercalcemia, depositing calcium in normal tissues
Metastatic calcification is a pathological process characterized by the deposition of calcium salts in normal, healthy tissues. This deposition is driven primarily by a state of persistent hypercalcemia, which can stem from excessive parathyroid hormone secretion, Vitamin D toxicosis, or bone resorption. The excess calcium precipitates out of the blood and deposits in alkaline microenvironments.

186. (C) Chronic renal failure causing secondary hyperparathyroidism
Chronic renal failure often leads to secondary hyperparathyroidism, where impaired phosphate excretion and decreased active Vitamin

D synthesis trigger excessive parathyroid hormone secretion. The resulting chronic hyperparathyroidism induces persistent hypercalcemia and hyperphosphatemia. This elevated calcium-phosphate product promotes the systemic deposition of calcium salts in healthy tissues, leading directly to metastatic calcification.

187. (D) Gangrene
Gangrene is a clinical term applied to a substantial area of necrotic tissue, usually on an extremity, that has undergone secondary invasion and digestion by putrefactive saprophytic bacteria. These bacteria break down cellular components, producing distinctive gas, dark discoloration, and a foul odor, which distinguishes gangrenous tissue from uncomplicated ischemic necrosis.

188. (A) Chronic ischemia causing coagulative necrosis and desiccation
Dry gangrene is a pathological condition that typically occurs on the body's extremities, such as the ears, tail, or distal limbs. It is initiated by chronic ischemia, which leads to coagulative necrosis of the tissue. Because the blood supply is limited, the affected tissue undergoes rapid desiccation and mummification before substantial bacterial putrefaction can occur.

189. (B) Programmed, energy-dependent cell death without surrounding inflammation
Apoptosis is a highly regulated, active form of programmed cell death that requires metabolic energy to proceed. It plays a vital role in removing unwanted or damaged cells without causing damage to surrounding tissues. Unlike necrosis, apoptosis does not trigger a local inflammatory response because the cell membrane remains intact, preventing the leakage of toxic contents.

190. (B) Formation of apoptotic bodies, which are phagocytosed by adjacent cells
During apoptosis, the dying cell undergoes characteristic structural changes, including cell shrinkage, chromatin condensation, and membrane blebbing. The cell eventually fragments into membrane-bound vesicles known as apoptotic bodies, which contain intact organelles and nuclear fragments. These structures are rapidly recognized and phagocytosed by adjacent cells or macrophages, avoiding an inflammatory response.

191. (A) The liver
Steatosis, or fatty change, is the abnormal accumulation of triglycerides within parenchymal cells. It occurs most frequently in the liver because this organ plays a central role in lipid metabolism, processing fatty acids from the diet and adipose tissue. Impairments in lipid uptake, oxidation, or lipoprotein secretion can quickly result in visible intracellular lipid vacuoles.

192. (C) Extracellular accumulation of insoluble, misfolded fibrillar proteins
Amyloidosis is a pathological condition characterized by the extracellular accumulation of insoluble, misfolded fibrillar proteins. These abnormal proteins aggregate into a distinctive beta-pleated sheet conformation, which resists enzymatic degradation. As these fibrils deposit in various tissues and organs, they disrupt normal tissue architecture and compromise organ function, often leading to clinical disease.

193. (D) Enlarged, pale, firm, and waxy
Grossly, organs heavily affected by severe amyloidosis undergo characteristic physical changes due to the extensive deposition of insoluble protein fibrils. The affected tissue typically appears significantly enlarged, pale, firm, and has a distinct waxy or translucent texture. These changes alter the organ's normal architecture and impair its physiological function, indicating advanced disease.

194. (C) Albinism
Albinism is a hereditary disorder characterized by a complete or partial lack of melanin production in the skin, hair, and eyes. It is caused by a genetic mutation that results in a functional deficiency of the enzyme tyrosinase, which is essential for synthesizing melanin from tyrosine. This condition increases susceptibility to solar radiation damage.

195. (B) Intracellular storage of iron derived from hemoglobin breakdown
Hemosiderin is a golden-yellow to golden-brown, granular, intracellular pigment derived from the breakdown of hemoglobin. It represents a stored, aggregated form of iron within macrophages and parenchymal cells, particularly following the phagocytosis of senescent or damaged erythrocytes. The pigment can be histologically identified and distinguished from other pigments using a Prussian blue stain.

Downloaded from www.pdf.tube

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

196. (D) Lipofuscin
Lipofuscin is known as the wear-and-tear or aging pigment. It consists of insoluble complexes of lipids and proteins that accumulate within the lysosomes of long-lived, post-mitotic cells, such as neurons and cardiac myocytes, over time. It is a product of the slow, incomplete peroxidation of autophagocytosed cellular membranes and serves as a marker of cellular aging.

197. (A) Carbon particles
Anthracosis is a common, non-pathogenic form of pneumoconiosis observed in animals residing in polluted urban or industrial environments. It is characterized by the accumulation of inhaled carbon or coal dust particles within alveolar macrophages and regional lymph nodes. These inert carbon deposits appear histologically as black, granular, intracellular material that does not provoke a significant inflammatory reaction.

198. (C) Infarct
An infarct is defined as a localized area of ischemic necrosis within a tissue or organ. This pathological lesion is caused by a sudden obstruction of either its arterial blood supply or its venous drainage. The occlusion leads to severe hypoxia, nutrient deprivation, and subsequent coagulative necrosis of the affected cells, typically producing a wedge-shaped gross appearance.

199. (A) Embolus
An embolus is a detached, intravascular solid, liquid, or gaseous mass that is transported by the bloodstream to a site distant from its point of origin. When it lodges in a vessel with a smaller lumen, it partially or completely occludes blood flow, potentially leading to local tissue ischemia, hypoxia, and subsequent infarction in the affected area.

200. (D) An active process characterized by increased arterial blood flow to a tissue
Hyperemia is an active physiological or pathological process characterized by an increased volume of blood within a tissue or organ. This condition is driven by active arteriolar dilation, which significantly increases arterial blood flow into the local microvasculature. Consequently, the affected tissue appears bright red, warm to the touch, and is typically associated with inflammation or active metabolic states.

201. (B) Decreased venous outflow, causing hypoxia, cyanosis, and dark discoloration
Congestion is a passive process resulting from impaired venous outflow from a tissue, whereas active hyperemia is driven by increased arterial inflow. In congestion, the accumulation of deoxygenated blood within the microvasculature causes tissue hypoxia, cyanosis, and a dark blue or reddish-blue discoloration. It is often associated with local venous obstruction or systemic heart failure.

NODIA

202. (C) Nutmeg liver
Chronic passive congestion of the liver, typically caused by right-sided heart failure, leads to a characteristic macroscopic pattern known as nutmeg liver. The obstruction of venous outflow results in severe congestion and necrosis of hepatocytes surrounding the central veins. This red-brown centrilobular congestion contrasts with the uncongested, pale, fatty periportal hepatocytes, mimicking the appearance of a sliced nutmeg.

203. (D) Edema
Edema is defined as the abnormal and excessive accumulation of fluid within the interstitial tissue spaces or extracellular body cavities. This pathological state arises when the physiological balance governing fluid movement between the vascular space and the interstitium is disrupted. It can present as localized swelling or as a generalized, systemic accumulation of fluid throughout the body.

204. (A) Increased capillary endothelial permeability
The formation of edema is governed by several primary pathophysiological mechanisms. Increased capillary endothelial permeability, which occurs during acute inflammation or tissue injury, allows plasma proteins and fluids to escape easily into the interstitium. This alteration disrupts normal osmotic gradients, leading to localized fluid accumulation that exceeds the drainage capacity of the lymphatic system.

205. (B) A profound decrease in intravascular colloid osmotic pressure
Hypoproteinemia, resulting from severe gastrointestinal parasitism, liver failure, or starvation, leads to a profound decrease in intravascular colloid osmotic pressure. Because albumin is the primary protein responsible

for maintaining this osmotic gradient, its depletion impairs the vascular system's ability to retain fluid. Consequently, fluid leaks into the interstitial spaces, causing generalized edema.

206. (B) Anasarca
Anasarca is a clinical and pathological term used to describe severe, generalized edema. It is characterized by widespread and profound fluid accumulation within the subcutaneous tissues throughout the entire body, often accompanied by fluid accumulation in major body cavities. This condition is commonly associated with severe renal failure, hepatic dysfunction, or congestive heart failure.

207. (A) Highly protein-rich with a high specific gravity and numerous cells
Exudate is an inflammatory fluid that is fundamentally distinguished from transudate by its high protein concentration, high specific gravity, and the presence of numerous inflammatory cells and cellular debris. It forms because inflammatory mediators increase vascular permeability, allowing large proteins and leukocytes to escape the microvasculature into the surrounding tissue spaces.

208. (C) Petechiae
Hemorrhage represents the escape of blood from the cardiovascular system due to vascular rupture or dysfunction. Petechiae are defined as minute, pinpoint hemorrhages measuring approximately one to two millimeters in diameter. They are commonly observed on the skin, mucosal membranes, or serosal surfaces and are typically associated with thrombocytopenia, platelet dysfunction, or vascular injury.

209. (D) Hematoma
A hematoma is a localized, massive accumulation of extravasated blood within a tissue space or body cavity. It is characterized by the formation of a palpable swelling or coagulated mass, which can compress adjacent structures and cause localized pain. This lesion typically results from the rupture of larger blood vessels due to trauma, surgery, or coagulopathy.

210. (C) Thrombosis
Thrombosis is a pathological process characterized by the formation of a solid or semi-solid mass of blood constituents within the unbroken cardiovascular system of a living animal. This event is driven by a cascade of vascular, cellular, and humoral factors. The resulting mass, or thrombus, can obstruct local blood flow, leading to ischemia or tissue infarction.

****

Click Here to Buy book on Amazon

CHAPTER 02

Haemodynamics Derangements, Degeneration and Necrosis

2006

1. Which of the following forces primarily opposes the filtration of fluid out of the capillary bed at the arterial end?
(A) Capillary hydrostatic pressure
(B) Interstitial fluid hydrostatic pressure
(C) Interstitial fluid colloid osmotic pressure
(D) Plasma colloid osmotic pressure

2001

2. A defining pathophysiological feature of an exudate, as opposed to a transudate, is its:
(A) Low specific gravity typically below 1.012
(B) Formation solely due to increased hydrostatic pressure
(C) High protein content with significant cellular debris
(D) Absence of inflammatory mediators

2025

3. In the pathogenesis of chronic passive congestion of the liver (nutmeg liver), which vascular alteration occurs first?
(A) Centrilobular necrosis of hepatocytes
(B) Dilation of central veins and sinusoids
(C) Periportal fatty change of hepatocytes
(D) Proliferation of tissue Kupffer cells

2021

4. Ischaemia causes hypoxic cellular injury primarily by compromising which critical intracellular process?
(A) Anaerobic glycolysis within the cytosol
(B) Protein synthesis by cellular ribosomes
(C) Lysosomal enzyme activation in cytoplasm
(D) Mitochondrial oxidative phosphorylation

2012

5. Which of the following best describes ecchymoses?
(A) Haemorrhages measuring 1 to 2 cm in diameter
(B) Minute 1 to 2 mm petechiae on skin surfaces
(C) Large accumulations of blood in body cavities
(D) Systemic bleeding from severe thrombocytopenia

2014

6. According to Virchow's triad, which condition is LEAST likely to predispose an individual to thrombosis?
(A) Damaged vascular endothelial cells
(B) Inherited hypercoagulable states
(C) Increased laminar blood flow rate
(D) Localized stasis of blood flow

2015

7. A fat embolism is most frequently associated with which of the following clinical events?
(A) Severe fracture of long bones
(B) Intravenous injection of air
(C) Deep vein thrombosis
(D) Rupture of an atherosclerotic plaque

NODIA

2007

8. Red (haemorrhagic) infarcts typically occur in organs with which of the following characteristics?
(A) Solid organs with end-arterial flow
(B) Tissues undergoing chronic atrophy
(C) Organs with dense collagenous capsules
(D) Tissues featuring dual blood supplies

2025

9. Sludged blood in the microvasculature is primarily characterized by the aggregation of erythrocytes due to:
(A) Increased systemic plasma albumin levels
(B) Plasma protein changes causing rouleaux
(C) Reduced vascular fibrinogen concentration
(D) Enhanced endothelial nitric oxide release

2008

10. Match the types of shock listed in List I with their primary underlying mechanisms listed in List II:
List I: (Types of Shock)
P. Cardiogenic shock
Q. Hypovolaemic shock
R. Septic shock
S. Anaphylactic shock
List II: (Mechanisms)

1. Systemic vasodilation and increased vascular permeability due to IgE-mediated response
2. Failure of the myocardial pump
3. Loss of blood or plasma volume
4. Peripheral vasodilation and pooling of blood due to microbial toxins

(A) P-2, Q-3, R-4, S-1
(B) P-2, Q-4, R-3, S-1
(C) P-3, Q-2, R-1, S-4
(D) P-4, Q-3, R-2, S-1

2010
11. Cellular swelling, a hallmark of reversible cell injury, is directly driven by the failure of which cellular component?
(A) Rough endoplasmic reticulum
(B) Golgi apparatus membranes
(C) Lysosomal membrane systems
(D) Sodium-potassium ATPase pump

2014
12. Hyaline change in tissue pathology is best classified as:
(A) A descriptive term for glassy, pink-staining material
(B) A specific biochemical accumulation of complex lipids
(C) An irreversible cellular degeneration specific to liver
(D) Extracellular deposit of calcium phosphate crystals

2018
13. Which of the following histochemical stains is most specific for identifying amyloid deposits in tissue sections?
(A) Hematoxylin and Eosin
(B) Congo Red
(C) Periodic Acid-Schiff
(D) Masson's Trichrome

2004
14. Which statement INCORRECTLY pairs a disturbance in lipid metabolism with its pathogenesis?
(A) Fatty change (steatosis) involves the abnormal accumulation of triglycerides within parenchymal cells.
(B) Niemann-Pick disease is caused by an excess of circulating low-density lipoproteins.
(C) Fatty infiltration refers to the accumulation of mature adipocytes in the stromal connective tissue.
(D) Steatosis is frequently observed in the liver due to its central role in fat metabolism.

2022
15. Choose the correct sequence of events leading to primary haemostasis after a vascular injury:
(A) Platelet adhesion → Vasoconstriction → Platelet aggregation → Fibrin deposition
(B) Vasoconstriction → Platelet adhesion → Platelet activation → Platelet aggregation
(C) Platelet activation → Vasoconstriction → Platelet adhesion → Fibrinolysis
(D) Vasoconstriction → Platelet aggregation → Platelet adhesion → Tissue factor release

2005
16. The accumulation of lipofuscin in myocardial cells is an indicator of which underlying process?
(A) Acute ischaemic necrosis
(B) Massive accumulation of exogenous carbon
(C) Previous free radical injury and aging
(D) Active viral myocarditis

2002
17. The pathogenesis of pre-hepatic (haemolytic) icterus is primarily characterized by an increase in which substance in the blood?
(A) Conjugated bilirubin
(B) Urobilinogen
(C) Biliverdin
(D) Unconjugated bilirubin

2019
18. Anthracosis is a benign pneumoconiosis primarily resulting from the inhalation and macrophage engulfment of which substance?
(A) Silica dust
(B) Asbestos fibers
(C) Carbon particles
(D) Iron oxide

2003
19. Dystrophic calcification occurs under which of the following physiological conditions?
(A) Hypercalcaemia and normal tissue integrity
(B) Normocalcaemia and areas of tissue necrosis
(C) Hyperparathyroidism and widespread tissue viability
(D) Vitamin D toxicity and normal tissue

2015
20. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).
Assertion (A): Apoptosis does NOT typically incite an inflammatory response in the surrounding tissue.
Reason (R): The plasma membrane of an apoptotic cell remains intact while being

Downloaded from www.pdf.tube

phagocytosed.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2012
21. Which morphological pattern of necrosis is most characteristic of hypoxic death of cells within the central nervous system?
(A) Liquefactive necrosis
(B) Coagulative necrosis
(C) Caseous necrosis
(D) Fibrinoid necrosis

2021
22. Dry gangrene is distinguished from moist gangrene by which of the following features?
(A) It features rapid progression with toxemia
(B) It typically results from venous occlusion
(C) It features a clear line of demarcation
(D) It exhibits extensive bacterial putrefaction

2025
23. During haemodynamic and metabolic stress, the earliest ultrastructural alteration observed in mitochondria is:
(A) Mitochondrial swelling and clearing of the matrix
(B) Rupture of the outer mitochondrial membrane
(C) Appearance of large, flocculent amorphous densities
(D) Complete dissolution of cristae

2021
24. Irreversible cell injury is most definitively indicated by which ultrastructural change involving lysosomes?
(A) Autophagosome formation in cytoplasm
(B) Mild swelling of the lysosomal matrix
(C) Accumulation of lipofuscin pigment
(D) Lysosomal rupture and enzyme leakage

2023
25. Which phenomenon best explains the movement of fluid from the interstitium back into the capillary at the venular end?
(A) Hydrostatic pressure exceeds colloid osmotic pressure.
(B) Colloid osmotic pressure exceeds hydrostatic pressure.
(C) Lymphatic drainage actively pumps fluid into the venule.
(D) Increased endothelial fenestrations allow passive diffusion.

2011
26. Which type of specific organ oedema is most directly lethal due to its interference with oxygen diffusion across alveolar septa?
(A) Cerebral oedema
(B) Dependent oedema
(C) Pulmonary oedema
(D) Periorbital oedema

2014
27. Heart failure cells found in the alveoli during chronic passive congestion of the lungs are essentially:
(A) Hypertrophied pneumocytes
(B) Erythrocytes containing inclusion bodies
(C) Degenerating neutrophils
(D) Haemosiderin-laden macrophages

2023
28. Which of the following is the most frequent aetiology of regional tissue ischaemia?
(A) Arterial thrombosis or embolism
(B) Generalized systemic hypotension
(C) Increased venous drainage
(D) Arteriolar vasodilation

2005
29. Purpura refers to haemorrhages that fall within which size range?
(A) Less than 1 mm
(B) 1 to 2 mm
(C) 3 to 10 mm
(D) Greater than 2 cm

NODIA

2014
30. Consider the following statements regarding the mechanisms of blood coagulation:
I. The intrinsic pathway is initiated by tissue factor (Factor III).
II. Thrombin converts soluble fibrinogen into insoluble fibrin.
III. Factor X activation represents the convergence of the intrinsic and extrinsic pathways.
Which combination represents the CORRECT statements?
(A) II and III
(B) I
(C) I and II
(D) III

2013
31. Systemic thromboembolism most frequently originates from thrombi located in the:
(A) Left heart chambers
(B) Pulmonary veins
(C) Right atrium
(D) Deep veins of the lower extremities

2022
32. White (anaemic) infarcts are characteristically found in which type of organ architecture?
(A) Spongy organs like the lungs
(B) Organs with extensive collateral circulation like the small intestine
(C) Solid organs with end-arterial circulation like the kidney
(D) Organs lacking a capsule

2017
33. The rheological phenomenon of sludged blood most directly leads to which physiological consequence?
(A) Increased arterial blood flow velocity
(B) Enhanced systemic oxygen gas delivery
(C) Rapid degradation of active red cells
(D) Decreased microvascular tissue perfusion

2017
34. In the compensatory stage of hypovolaemic shock, the body attempts to maintain blood pressure primarily through:
(A) Peripheral vasodilation
(B) Decreased heart rate
(C) Activation of the sympathetic nervous system
(D) Suppression of the renin-angiotensin-aldosterone system

2007
35. Ballooning degeneration in viral infections, such as poxvirus lesions, is a severe manifestation of which basic cellular process?
(A) Intracellular steatosis
(B) Hydropic cellular swelling
(C) Coagulative tissue necrosis
(D) Intracellular glycogen storage

2008
36. Which condition is caused by the deposition of urate crystals in the articular cartilage and periarticular tissues?
(A) Mucoid degeneration
(B) Primary amyloidosis
(C) Hyaline change
(D) Gout

2016
37. Which specific biochemical classification of amyloid is typically deposited in patients suffering from chronic inflammatory conditions?
(A) AA amyloid
(B) AL amyloid
(C) ATTR amyloid
(D) Aβ amyloid

2015
38. The pathogenesis of fatty change (steatosis) in the liver due to alcohol toxicity involves which primary metabolic derangement?
(A) Decreased esterification of fatty acids to triglycerides
(B) Increased oxidation of fatty acids in mitochondria
(C) Increased synthesis of triglycerides due to excess NADH
(D) Enhanced synthesis and release of lipoproteins

2015
39. Which genetic disorder is categorized as a glycogenosis (glycogen storage disease) affecting primarily the liver and kidneys?
(A) Von Gierke disease
(B) Niemann-Pick disease
(C) Tay-Sachs disease
(D) Gaucher disease

2015
40. Consider the following statements regarding endogenous pigment metabolism:
I. Melanin is produced by melanocytes through the oxidation of tyrosine.
II. Lipofuscin is an indicator of acute cellular injury.
III. Ceroid is an autofluorescent pigment similar to lipofuscin but often found in pathological states.
IV. Melanin is predominantly stored in lysosomes of neutrophils.
Which combination represents the CORRECT statements?
(A) I, II and IV
(B) I and III
(C) II, III and IV
(D) II and IV

2006
41. Haemosiderosis differs from haemochromatosis primarily in that haemosiderosis:
(A) Is associated with fibrosis and tissue damage
(B) Represents iron overload without tissue injury

Click Here to Buy book on Flipkart

(C) Involves copper accumulation rather than iron
(D) Represents an exclusively localized phenomenon

2004
42. Silicosis induces pulmonary pathology primarily through the activation and subsequent death of which cells?
(A) Type II pneumocytes
(B) Endothelial cells
(C) Alveolar macrophages
(D) Eosinophils

2006
43. Heterotopic ossification is best defined as the:
(A) Calcification of degenerating tumor tissues
(B) Decalcification of the skeleton due to hyperparathyroidism
(C) Deposition of urates in the joint capsule
(D) Formation of mature bone in non-osseous soft tissues

2019
44. The execution phase of apoptosis relies heavily on the activation of which family of proteases?
(A) Caspases
(B) Matrix metalloproteinases
(C) Cathepsins
(D) Elastases

2019
45. Arrange the following steps of the extrinsic pathway of apoptosis in the correct order:
I. Cleavage and activation of executioner caspases
II. FasL binds to Fas receptor
III. Activation of Caspase-8
IV. Formation of the Death-Inducing Signaling Complex (DISC)
(A) II, IV, III, I
(B) II, III, IV, I
(C) IV, II, III, I
(D) IV, III, II, I

2010
46. Caseous necrosis is histologically characterized by:
(A) Complete preservation of dead cell outlines
(B) Structureless, granular, cheese-like debris
(C) Transformation of tissue into liquid mass
(D) Deposition of immune complexes in vessels

2023
47. Gas gangrene is predominantly caused by infection with which genus of bacteria?
(A) Clostridium
(B) Staphylococcus
(C) Streptococcus
(D) Mycobacterium

2018
48. During metabolic stress, the dissociation of ribosomes from the rough endoplasmic reticulum directly results in:
(A) Enhanced lipid synthesis
(B) Accelerated oxidative phosphorylation
(C) Decreased cellular protein synthesis
(D) Immediate cellular lysis

2013
49. Irreversible damage to the plasma membrane during cell injury primarily causes:
(A) Mild cellular swelling and cytoplasm changes
(B) Increased transcription of cell survival genes
(C) Reversible plasma membrane blebbing formation
(D) Massive calcium influx and enzyme leakage

2010
50. Match the pathological pigments listed in List I with their underlying nature listed in List II:
List I: (Pathological Pigments)
P. Melanin
Q. Haemosiderin
R. Lipofuscin
S. Bilirubin
List II: (Nature)
1. Wear-and-tear lipid polymer
2. Iron-containing storage aggregate
3. Tyrosine-derived endogenous pigment
4. Porphyrin ring degradation product
(A) P-1, Q-2, R-3, S-4
(B) P-3, Q-2, R-1, S-4
(C) P-2, Q-3, R-4, S-1
(D) P-3, Q-1, R-2, S-4

2012
51. The primary cause of non-inflammatory oedema (transudate) in patients with severe right-sided heart failure is:
(A) Decreased plasma colloid osmotic pressure
(B) Increased capillary hydrostatic pressure
(C) Obstruction of local lymphatic drainage
(D) Increased microvascular wall permeability

2015
52. Which mechanism best explains the development of localized oedema following lymphatic obstruction (lymphoedema)?
(A) Excessive retention of sodium and water by kidneys

Downloaded from www.pdf.tube

(B) Sudden increase in intravascular hydrostatic pressure
(C) Overproduction of albumin proteins by the liver
(D) Failure to drain interstitial fluid and proteins

2012

53. Active hyperaemia is best described as:
(A) Increased arterial inflow of blood to tissues
(B) Impaired venous outflow causing blood accumulation
(C) Chronic pooling of blood in lower extremities
(D) Reduction in vascular blood flow from vasospasm

2022

54. The initial cellular response to hypoxia induced by ischaemia is:
(A) Rapid replication of cellular DNA
(B) Formation of autophagic vacuoles
(C) Depletion of intracellular ATP
(D) Hypertrophy of the Golgi apparatus

2005

55. Petechiae are typically associated with which of the following underlying defects?
(A) Platelet dysfunction
(B) Factor VIII deficiency
(C) Aortic aneurysm rupture
(D) Portal hypertension

2023

56. Which of the following is considered a primary (genetic) hypercoagulable state predisposing to thrombosis?
(A) Prolonged patient bed rest
(B) Disseminated systemic cancer
(C) Chronic atrial fibrillation
(D) Factor V Leiden mutation

2018

57. Which type of embolus typically occurs as a complication of decompression sickness (Caisson disease)?
(A) Amniotic fluid embolus
(B) Air (nitrogen) embolus
(C) Thromboembolus
(D) Fat embolus

2010

58. The morphological evolution of an infarct typically progresses through which sequence of inflammatory responses?
(A) Macrophage infiltration, followed by neutrophils, then fibrosis
(B) Granulation tissue formation followed by acute inflammation
(C) Necrosis, acute inflammation, scavenging, and granulation
(D) Lymphocytic infiltration followed directly by calcification

2003

59. Sludged blood exacerbates tissue hypoxia primarily by:
(A) Increasing oxygen-binding affinity of hemoglobin
(B) Increasing local venule hydrostatic pressure
(C) Directly causing acute arterial thrombosis
(D) Impairing perfusion and increasing viscosity

2006

60. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).
Assertion (A): Septic shock involves widespread peripheral vasodilation and increased vascular permeability.
Reason (R): Endotoxins trigger massive release of inflammatory cytokines, including TNF and IL-1, causing systemic endothelial activation.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2022

61. Hydropic degeneration is morphologically identifiable under the light microscope by the appearance of:
(A) Dark, shrunken nuclei with eosinophilic cytoplasm
(B) Clear cytoplasmic vacuoles from distended ER
(C) Intracytoplasmic crystalline mineral deposits
(D) Granular accumulation of wear-and-tear pigment

NODIA

2021

62. Mucoid/myxomatous degeneration is characterized by the overproduction of which substance in the connective tissue?
(A) Fibrinogen proteins
(B) Glycosaminoglycans
(C) Keratinized proteins
(D) Cholesterol crystals

2016

63. Amyloid deposition causes tissue damage primarily through:

(A) Pressure atrophy of adjacent functional cells
(B) Direct cytotoxic destruction of cell membranes
(C) Inciting a massive purulent inflammatory response
(D) Stimulating rapid cellular proliferation

2023
64. Lipid storage diseases (sphingolipidoses), such as Tay-Sachs disease, are caused by:
(A) Excessive chronic dietary intake of lipids
(B) Overactivity of lipid synthetic pathways
(C) Deficiency of specific lysosomal enzymes
(D) Autoimmune destruction of lipophilic cells

2004
65. In diabetes mellitus, abnormal carbohydrate metabolism can lead to the pathological accumulation of glycogen in the:
(A) Erythrocytes within the vascular system
(B) Stratum corneum cells of the epidermis
(C) Alveolar macrophages within the lungs
(D) Epithelium of the renal proximal tubules

2001
66. The brown pigment associated with wear-and-tear and cellular aging, composed of lipid-protein complexes, is:
(A) Haemosiderin
(B) Melanin
(C) Lipofuscin
(D) Bilirubin

2019
67. Porphyria is a group of metabolic disorders characterized by the defective biosynthesis of:
(A) Melanin
(B) Heme
(C) Glycogen
(D) Cholesterol

2022
68. Which occupational pneumoconiosis is characterized by the formation of dense collagenous nodules and presents a significantly increased risk of developing tuberculosis?
(A) Anthracosis
(B) Berylliosis
(C) Byssinosis
(D) Silicosis

2009
69. Metastatic calcification is distinguished from dystrophic calcification by which essential factor?
(A) It requires the presence of hypercalcaemia.
(B) It occurs in areas of previous tissue necrosis.
(C) It is localized primarily to damaged heart valves.
(D) It does not involve calcium phosphate deposition.

2011
70. Consider the following statements regarding necrosis:
I. Necrosis is accompanied by an inflammatory response.
II. Fibrinoid necrosis is characteristic of immune complex deposition in blood vessels.
III. Coagulative necrosis is typical of central nervous system infarcts.
Which combination represents the CORRECT statements?
(A) II and III
(B) I and II
(C) I and III
(D) I

2014
71. Fat necrosis is most commonly associated with acute trauma to adipose tissue or the pathological release of enzymes from the:
(A) Pancreas
(B) Liver
(C) Spleen
(D) Kidney

2013
72. Moist gangrene is clinically more dangerous than dry gangrene because it is characterized by:
(A) Slow disease progression over months or years
(B) Presence of a distinct line of demarcation
(C) Occurrence limited strictly to cold exposure
(D) Rapid spread with putrefaction and toxemia

2014
73. During early reversible cell injury, the ultrastructural dilation of the endoplasmic reticulum directly correlates with:
(A) The breakdown of nuclear chromatin
(B) The detachment of ribosomes
(C) The synthesis of new mitochondria
(D) The formation of lipid vacuoles

2023
74. Which cytoskeletal alteration is commonly seen in irreversible cell injury?
(A) Enhanced polymerization of actin and myosin filaments
(B) Formation of hyperstable, rigid microtubule arrays
(C) Breakdown of filaments leading to membrane

Click Here to Buy book on Amazon

fragmentation
(D) Increased synthesis of intermediate structural filaments

2010
75. The physiological condition of Starling forces is best represented by the equation:
(A) $J_v = K_f[(P_c + P_i) - (\pi_c + \pi_i)]$
(B) $J_v = K_f[(\pi_c - \pi_i) - (P_c - P_i)]$
(C) $J_v = K_f[(P_i - P_c) + (\pi_c - \pi_i)]$
(D) $J_v = K_f[(P_c - P_i) - (\pi_c - \pi_i)]$

2011
76. An exudate is fundamentally the result of which underlying pathophysiological mechanism?
(A) Increased microvascular permeability
(B) Increased capillary hydrostatic pressure
(C) Decreased plasma albumin concentration
(D) Obstruction of local lymphatic vessels

2010
77. The gross appearance of a "nutmeg liver" results from alternating areas of:
(A) Amyloid deposition and normal parenchyma
(B) Centrilobular congestion and fatty change
(C) Bile accumulation and widespread fibrosis
(D) Haemorrhage and pale tumor infiltrates

2002
78. Which sequence correctly describes the cellular consequences of ischaemia?
(A) Loss of ATP → Na^+/K^+ pump failure → Cellular swelling
(B) Na^+/K^+ pump failure → ATP loss → Membrane rupture
(C) Cellular swelling → Loss of ATP → Ribosomal detachment
(D) Lysosomal rupture → ATP loss → Apoptosis

2012
79. Haemostasis involves the conversion of soluble fibrinogen to an insoluble fibrin polymer. This step is directly catalyzed by:
(A) Thrombin
(B) Plasmin
(C) Tissue factor
(D) Factor XII

2011
80. Consider the following statements regarding infarction:
I. Red infarcts happen only in solid organs with end-arterial supply.
II. White infarcts occur in spongy organs like the lungs.
III. An infarct results from the occlusion of either arterial supply or venous drainage.
IV. Infarcts generally undergo coagulative necrosis, except in the brain.
Which combination represents the INCORRECT statements?
(A) I and II
(B) III and IV
(C) I, II and III
(D) I, III and IV

2025
81. Parasitic emboli in veterinary pathology are most classically associated with the migration of which parasite in horses?
(A) Fasciola hepatica
(B) Dirofilaria immitis
(C) Toxocara canis
(D) Strongylus vulgaris

2025
82. Infarcts are characteristically wedge-shaped. The apex of this wedge typically points toward the:
(A) Capsule of the organ
(B) Center of the necrotic zone
(C) Occluded blood vessel
(D) Main venous drainage

2008
83. The aggregation of red blood cells in sludged blood is typically driven by:
(A) High levels of intracellular energy stores
(B) Reduced surface charge from plasma proteins
(C) Increased velocity of microvascular flow
(D) Destruction of endothelial glycocalyx layer

2006
84. Which clinical stage of shock is characterized by tissue hypoxia, anaerobic glycolysis, lactic acidosis, and blunting of the vasomotor response?
(A) Nonprogressive (compensatory) stage
(B) Irreversible stage
(C) Prodromal stage
(D) Progressive stage

NODIA

2016
85. Cellular swelling is deemed reversible because:
(A) The membrane is intact and injury can revert
(B) Mitochondrial dense bodies have already formed
(C) Nuclear karyorrhexis has initiated and stopped
(D) Lysosomal enzymes have degraded the cytoskeleton

2024
86. Gout is characterized by the tissue deposition of:
(A) Calcium pyrophosphate
(B) Cholesterol crystals
(C) Monosodium urate
(D) Amyloid light chains

2012
87. In amyloidosis, the secondary (reactive) form is predominantly composed of AA amyloid. This protein is synthesized by the liver derived from which acute-phase reactant?
(A) Serum amyloid-associated protein
(B) Serum amyloid P component protein
(C) Circulating C-reactive protein
(D) Immunoglobulin light chain protein

2009
88. A common histopathological feature of steatosis in the liver is the presence of:
(A) Extracellular lipid pools surrounded by giant cells
(B) Intranuclear lipid inclusions within hepatocytes
(C) Hyperplastic Kupffer cells containing haemosiderin
(D) Cytoplasmic lipid vacuoles displacing the nucleus

2004
89. Glycogenoses (glycogen storage diseases) represent disturbances of carbohydrate metabolism caused by:
(A) Chronic sustained hyperglycemia in tissues
(B) Inherited deficiencies of glycogen enzymes
(C) Autoimmune destruction of pancreatic beta cells
(D) Defective insulin receptors on skeletal muscle

2001
90. Match the types of necrosis listed in List I with their typical clinical or pathological contexts listed in List II:
List I: (Types of Necrosis)
P. Coagulative necrosis
Q. Liquefactive necrosis
R. Caseous necrosis
S. Fat necrosis
List II: (Clinical or Pathological Contexts)
1. Acute pancreatitis
2. Tuberculosis infection
3. Myocardial infarction
4. Cerebral infarction
(A) P-3, Q-4, R-2, S-1
(B) P-4, Q-3, R-2, S-1
(C) P-3, Q-4, R-1, S-2
(D) P-2, Q-4, R-3, S-1

2024
91. Icterus (jaundice) results from the accumulation of bilirubin. Post-hepatic icterus is primarily caused by:
(A) Excessive breakdown of erythrocytes
(B) Hepatocellular injury and failure of conjugation
(C) Genetic defect in bilirubin uptake
(D) Obstruction of the biliary tree

2004
92. Which exogenous pigment, heavily associated with the inhalation of coal dust, causes black discoloration of the lungs and regional lymph nodes?
(A) Carbon dust
(B) Silica dust
(C) Asbestos dust
(D) Iron dust

2010
93. A classic location for dystrophic calcification is:
(A) Gastric mucosa in hypercalcaemic states
(B) Atherosclerotic plaques in large arteries
(C) Healthy renal proximal tubules in kidneys
(D) Alveolar walls in metastatic lung disease

2001
94. Apoptotic bodies are distinct from necrotic debris because they:
(A) Contain randomly degraded, smeared nuclear DNA
(B) Are membrane-bound and do not leak contents
(C) Induce a robust, acute neutrophilic infiltrate
(D) Represent swollen and ruptured cell organelles

2017
95. Fibrinoid necrosis is classically identified in the walls of:
(A) Blood vessels
(B) Large airways
(C) The gastrointestinal tract
(D) The ventricular myocardium

2012
96. Which feature most reliably differentiates gas gangrene from moist gangrene?
(A) Presence of active putrefactive bacteria
(B) Rapid progression of gangrene in limbs
(C) Crepitus from bacterial gas production
(D) Direct involvement of the internal organs

Downloaded from www.pdf.tube

2011

97. Ultrastructurally, the "myelin figures" frequently seen in injured cells represent:
(A) Aggregated ribosomes in the cytoplasm
(B) Viral inclusion bodies within the nucleus
(C) Clusters of swollen functional mitochondria
(D) Whorls of lipids from damaged cell membranes

2012

98. The point of no return in irreversible cell injury is most closely associated with:
(A) Mild cellular swelling and cytoplasm changes
(B) Detachment of ribosomes from the rough ER
(C) Severe mitochondrial and membrane dysfunction
(D) Clumping of nuclear chromatin in the nucleus

2011

99. The most significant factor maintaining the intravascular colloidal osmotic pressure is plasma:
(A) Fibrinogen
(B) Albumin
(C) Globulin
(D) Prothrombin

2002

100. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).
Assertion (A): Ischaemia is a more rapid and severe cause of cellular injury than simple hypoxia.
Reason (R): Ischaemia halts the delivery of substrates for glycolysis and prevents the removal of toxic metabolic byproducts.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2010

101. Passive congestion results from:
(A) Impaired venous drainage from a tissue
(B) Increased arterial vasodilation
(C) Increased metabolic demand
(D) Sympathetic nervous system overactivity

2005

102. An early morphologic hallmark of ischemic cell injury under the light microscope is:
(A) Karyolysis and chromatin clumping
(B) Extensive tissue fibrosis deposition
(C) Eosinophilia and loss of cell detail
(D) Massive neutrophilic cell infiltration

2008

103. In the process of haemostasis, von Willebrand factor (vWF) primarily acts as a bridge between:
(A) Platelets and subendothelial collagen
(B) Thrombin molecules and fibrinogen proteins
(C) Fibrin monomers during cross-link formation
(D) Platelets and circulating neutrophilic cells

2009

104. The pathogenesis of thrombosis is heavily influenced by endothelial injury. Which mechanism is a direct pro-coagulant effect of injured endothelium?
(A) Release of vasodilatory nitric oxide
(B) Secretion of anti-platelet prostacyclin
(C) Upregulation of surface thrombomodulin
(D) Exposure of tissue factor and matrix

2009

105. Choose the correct sequence of the classical coagulation cascade (extrinsic to common pathway):
(A) Tissue Factor → Factor Xa → Thrombin → Fibrin
(B) Factor XII → Factor Xa → Thrombin → Fibrin
(C) Thrombin → Factor Xa → Tissue Factor → Fibrin
(D) Fibrin → Thrombin → Factor Xa → Tissue Factor

2008

106. Paradoxical embolism refers to an embolus that:
(A) Arises in the systemic arterial system and lodges within a distal arterial bed
(B) Originates in the venous system and enters systemic arteries via a cardiac defect
(C) Travels in a retrograde direction against the normal physiological flow of blood
(D) Disintegrates spontaneously via endogenous fibrinolytic pathways prior to occlusion

NODIA

2007

107. Which finding makes a hemorrhagic (red) infarct more likely than a white infarct?
(A) Reperfusion of previously ischemic tissue
(B) Dense tissue structure
(C) Complete arterial occlusion in an end-artery system
(D) Lack of collateral blood vessels

2024

108. Sludged blood primarily impacts tissue health by:
(A) Increasing physiological oxygen diffusion distance across tissue beds
(B) Directly obstructing large-caliber elastic and muscular arteries
(C) Inducing capillary stasis and widespread microvascular thrombosis
(D) Elevating systemic arterial blood pressure throughout the vascular tree

2021

109. Anaphylactic shock is characterized pathophysiologically by:
(A) Myocardial injury causing a profound reduction in systemic contractility
(B) Rapid and severe depletion of circulating intravascular blood volume
(C) Systemic endotoxin release during severe gram-negative septicaemia
(D) IgE-mediated mast cell degranulation triggering systemic vasodilation

2010

110. Consider the following statements regarding reversible cell injury:
I. It is characterized by the formation of myelin figures.
II. It invariably involves severe mitochondrial vacuolization and rupture.
III. Cellular swelling (hydropic change) is a universal early feature.
Which of the above statements is/are correct?
(A) I and II
(B) III
(C) I and III
(D) II

2007

111. Russell bodies found in plasma cells are an example of which type of metabolic disturbance?
(A) An inherited systemic glycogen storage pathway defect
(B) Abnormal intracellular accumulation of folded proteins
(C) Severe hepatocellular lipid deposition and fatty change
(D) Pathological deposition of complex endogenous pigments

2005

112. Amyloid proteins share a common physical structural property, which is:
(A) Predominance of alpha-helical protein domains
(B) Spontaneous random coil polypeptide formation
(C) Conserved triple-helical collagenous structures
(D) Non-branching beta-pleated sheet conformation

2001

113. Macrophages that have phagocytosed excessive amounts of lipid in tissues are commonly referred to as:
(A) Foam cells
(B) Langhans giant cells
(C) Epithelioid cells
(D) Heart failure cells

2009

114. Pompe disease is a glycogenosis characterized by the lack of lysosomal acid maltase, primarily causing severe accumulation of glycogen in:
(A) Neurons of the central and peripheral nervous system
(B) Hepatocytes located within the hepatic parenchyma
(C) Myofibers of the cardiac and skeletal muscle systems
(D) Macrophage populations within the splenic parenchyma

2022

115. Which biochemical marker is most definitive for detecting apoptosis before morphological changes are overt?
(A) Caspase 3 activation
(B) Elevated lactate dehydrogenase (LDH)
(C) Increased ATP levels
(D) Formation of myelin figures

2013

116. Haemosiderin is primarily composed of:
(A) Oxidized melanin
(B) Degraded porphyrin rings lacking iron
(C) Lipoprotein complexes
(D) Aggregates of ferritin micelles

2023

117. Asbestos exposure, beyond causing asbestosis, is specifically linked to an increased risk of which malignant neoplasm?
(A) Hepatocellular carcinoma
(B) Malignant mesothelioma
(C) Renal cell carcinoma
(D) Melanoma

Click Here to Buy book on Flipkart

2005
118. Which statement regarding metastatic calcification is LEAST relevant to its pathogenesis?
(A) It requires elevated systemic calcium or phosphate levels.
(B) It commonly deposits in the interstitial tissues of the gastric mucosa, kidneys, and lungs.
(C) It is driven by the necrosis of underlying tissues.
(D) It can be caused by hyperparathyroidism.

2019
119. A hallmark biochemical change unique to the execution phase of apoptosis is:
(A) Random, diffuse enzymatic cleavage of genomic DNA throughout the entire nucleus
(B) Massive, rapid depletion of intracellular adenosine triphosphate within cytoplasm
(C) Widespread denaturation of all structural and functional intracellular proteins
(D) Internucleosomal DNA cleavage yielding fragments in multiples of ~200 base pairs

2008
120. Consider the following statements about necrosis:
I. Coagulative necrosis preserves tissue architecture for a few days.
II. Liquefactive necrosis is associated with bacterial or fungal infections.
III. Caseous necrosis is exclusively found in acute viral infections.
IV. Fat necrosis always results from autoimmune disease.
Which of the above statements is/are correct?
(A) I and II
(B) II, III and IV
(C) I, II and III
(D) III and IV

2008
121. Dry gangrene occurs almost exclusively in which anatomical location?
(A) The alveolar parenchymal tissue of the lungs
(B) The distal portions of the lower extremities
(C) The mucosal lining of the small intestines
(D) The parenchymal vascular tissue of the liver

2008
122. During metabolic stress, which change directly reflects a decrease in the energy state of the cell?
(A) Increased glycogen stores
(B) Loss of mitochondrial matrix granules
(C) Swelling of the endoplasmic reticulum
(D) Proliferation of smooth endoplasmic reticulum

2002
123. The transition from reversible to irreversible cell injury is marked ultrastructurally by:
(A) Amorphous densities within the mitochondrial matrix
(B) Generalized mild swelling of the endoplasmic reticulum
(C) Dense clumping of chromatin along the nuclear envelope
(D) Formation of transient blebs on the cellular membrane

2004
124. Starling forces balance fluid movement. If plasma colloid osmotic pressure decreases, the net result will be:
(A) Net movement of fluid back into the capillary lumen
(B) Compensatory reduction in lymphatic system drainage
(C) Movement of fluid into the interstitium causing edema
(D) Significant elevation of local hydrostatic pressure

2018
125. The specific gravity of an exudate is generally:
(A) Less than 1.012
(B) Exactly 1.000
(C) Lower than that of a transudate
(D) Greater than 1.020

2013
126. Active hyperaemia in skeletal muscle during exercise is mediated primarily by:
(A) Mechanical obstruction of draining venules and veins
(B) Systemic activation of parasympathetic neural tone
(C) Local release of nitric oxide and adenosine molecules
(D) Anatomical blockage of regional lymphatic channels

NODIA

2019
127. Which type of cell is most susceptible to hypoxic-ischaemic injury?
(A) Skeletal muscle cells
(B) Neurons
(C) Fibroblasts
(D) Epidermal cells

2006
128. A key mechanism of haemostasis involves platelet aggregation. Which molecule directly binds platelets together during this process?
(A) Tissue factor
(B) Plasminogen
(C) Prostacyclin
(D) Fibrinogen

2013
129. Virchow's triad encompasses all of the following EXCEPT:
(A) Decreased capillary hydrostatic pressure
(B) Acute or chronic endothelial cell injury
(C) Turbulent or stasis-induced blood flow
(D) Inherited or acquired hypercoagulability

2012
130. Match the metabolic disturbance with its hallmark manifestation:
List I: (Metabolic Disturbance)
P. Protein metabolism
Q. Lipid metabolism
R. Carbohydrate metabolism
S. Uric acid metabolism
List II: (Manifestation)
1. Glycogenoses
2. Gout
3. Hyaline change
4. Steatosis
(A) P-3, Q-4, R-1, S-2
(B) P-4, Q-3, R-1, S-2
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-3, S-2

2002
131. The term 'lines of Zahn' is morphologically diagnostic of:
(A) Thrombi formed in flowing blood
(B) Clots formed after somatic death
(C) Areas of infarcted cardiac muscle
(D) Sludged blood within capillaries

2018
132. Bone marrow embolism is most typically observed following:
(A) Accidental displacement of indwelling venous catheters
(B) Extensive thermal injury of subcutaneous soft tissues
(C) Widespread disseminated intravascular coagulation state
(D) Cardiopulmonary resuscitation resulting in rib fractures

2010
133. Which cellular adaptation or injury is LEAST likely to result from chronic, sublethal ischaemia?
(A) Cellular atrophy
(B) Coagulative necrosis
(C) Fatty change
(D) Decreased cell size

2008
134. Which phenomenon directly contributes to the rheological changes seen in sludged blood?
(A) Decreased fibrinogen levels
(B) Massive leukocyte rolling
(C) Erythrocyte rouleaux formation
(D) Enhanced platelet degranulation

2008
135. Arrange the events of intrinsic pathway apoptosis in the correct chronological order:
I. Cytochrome c release from mitochondria
II. Formation of the apoptosome
III. Activation of Caspase-9
IV. Activation of executioner caspases (e.g., Caspase-3)
(A) I, II, III, IV
(B) I, III, II, IV
(C) II, I, III, IV
(D) III, I, II, IV

2002
136. The irreversible stage of shock is defined physiologically by:
(A) Cellular injury causing multi-organ failure despite resuscitation
(B) Reversible decline in blood pressure managed with fluid therapies
(C) Widespread shift to anaerobic glycolysis within parenchymal cells
(D) Maximum compensatory activation of systemic sympathetic reflexes

2013
137. Which term describes the severe vacuolar degeneration observed in the renal tubular epithelium following exposure to osmotic agents or severe hypokalemia?
(A) Fatty change
(B) Hydropic degeneration
(C) Amyloidosis
(D) Fibrinoid change

NODIA

2023
138. Which condition is a well-known consequence of disturbances in purine metabolism?
(A) Porphyria
(B) Gout

(C) Glycogen storage disease
(D) Amyloidosis

2003

139. Which organ is classically described as having a "sago" or "lardaceous" appearance when extensively infiltrated with amyloid?
(A) Spleen
(B) Kidney
(C) Liver
(D) Heart

2025

140. Identify the correct option regarding the Assertion (A) and Reason (R) given below:
Assertion (A): Lipofuscin pigment is considered an innocuous byproduct of cellular aging.
Reason (R): Lipofuscin actively promotes cellular necrosis by disrupting lysosomal membranes.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2017

141. The abnormal accumulation of triglycerides in the liver due to starvation is primarily caused by:
(A) Impaired hepatic synthesis of systemic apoproteins
(B) Decreased mitochondrial oxidation of active fatty acids
(C) Diminished mobilization of lipid from adipose tissues
(D) Excessive mobilization of peripheral free fatty acids

2005

142. Diabetic microangiopathy involves the thickening of basement membranes, which is fundamentally a disturbance of which metabolic pathway?
(A) Lipid metabolism
(B) Protein metabolism
(C) Carbohydrate metabolism
(D) Endogenous pigment metabolism

2009

143. Melanin is synthesized from tyrosine by the enzyme tyrosinase. A complete genetic deficiency of this enzyme results in:
(A) Melanoma
(B) Albinism
(C) Vitiligo
(D) Ochronosis

2013

144. Which statement best defines porphyria?
(A) An acquired defect in systemic cellular iron storage
(B) A disease causing excessive production of bilirubin
(C) A failure of the liver to accept conjugated bilirubin
(D) An enzymatic defect in the heme biosynthetic pathway

2022

145. Byssinosis is a pneumoconiosis associated with the inhalation of:
(A) Cotton dust
(B) Silica dust
(C) Asbestos fibers
(D) Iron particles

2011

146. Which fundamental process initiates dystrophic calcification in necrotic tissue?
(A) Systemic hypercalcemic states leading to vascular deposition
(B) Excessive endocrine overproduction of parathyroid hormone
(C) Mitochondrial calcium entry and binding to denatured proteins
(D) Pathological osteoblast activation within injured soft tissues

2008

147. Which gene acts as a primary pro-apoptotic regulator by promoting mitochondrial outer membrane permeabilization?
(A) Bax
(B) Bcl-2
(C) Bcl-xL
(D) c-Myc

2001

148. The microscopic appearance of coagulative necrosis reveals:
(A) Complete loss of cellular outlines with a prominent neutrophilic infiltrate
(B) Amorphous granular debris without any cellular ghosts
(C) Liquefaction of the affected area
(D) Preservation of the cellular outline but loss of nuclear details

2025

149. Moist gangrene of the intestine most frequently results from:
(A) Exposure of tissues to extreme environmental frostbite

Downloaded from www.pdf.tube

(B) Volvulus or intussusception causing vascular occlusion
(C) Chronic arterial atherosclerosis without active infection
(D) Superficial bacterial infections involving the epidermis

2013
150. Consider the following statements about thrombosis and haemostasis:
I. Thromboxane A_2 promotes platelet aggregation.
II. Prostacyclin (PGI_2) inhibits platelet aggregation.
III. Plasmin degrades fibrin into D-dimers.
IV. Endothelial cells synthesize von Willebrand factor.
V. Antithrombin III directly activates thrombin.
Which of the above statements is/are correct?
(A) I, II, III and IV
(B) I, II and III
(C) II, III, IV and V
(D) I, III and V

2012
151. During necrosis, the progressive enzymatic dissolution of the cell nucleus, where chromatin fades and eventually disappears, is termed:
(A) Pyknosis
(B) Karyolysis
(C) Karyorrhexis
(D) Chromatolysis

2021
152. Which mechanism prevents fluid from excessively accumulating in the normal interstitial space?
(A) Continuous fluid drainage by the lymphatic system
(B) Complete capillary wall impermeability to water
(C) Maintenance of a high interstitial oncotic pressure
(D) Active cellular transport of water by fibroblasts

2011
153. A 6-year-old dog presents with right-sided heart failure. Pathophysiologically, you would most expect to find:
(A) Widespread alveolar flooding and pulmonary edema
(B) Severe ascites and chronic hepatic congestion
(C) Progressive left atrial and ventricular dilation
(D) Acute interstitial cerebral edema in the brain

NODIA

2019
154. Hypoxia specifically causes an increase in which metabolic byproduct, leading to lowered intracellular pH?
(A) Urea
(B) Lactic acid
(C) Bicarbonate
(D) Ammonia

2009
155. Petechial haemorrhages on the mucosal surfaces are most strongly indicative of:
(A) Primary haemostatic defect such as thrombocytopenia
(B) Coagulation factor deficiency such as haemophilia
(C) Severe blunt trauma causing deep muscular contusion
(D) Ruptured aneurysm within the cerebral circulation

2025
156. The intrinsic pathway of blood coagulation is triggered in vitro by:
(A) Release of tissue factor from damaged vascular cells
(B) Enzymatic activation of circulating serum plasminogen
(C) Exposure of factor XII to a negatively charged surface
(D) Binding of von Willebrand factor to active platelets

2014
157. A thrombus formed in the deep veins of the leg (DVT) is most likely to embolize and lodge in the:
(A) Brain
(B) Coronary arteries
(C) Renal arteries
(D) Pulmonary arteries

2018
158. The white/anaemic appearance of an infarct in the kidney is primarily due to:
(A) Massive infiltration of neutrophils and other inflammatory leukocytes
(B) Rapid dystrophic calcification occurring within the necrotic tissue
(C) Lack of collateral blood flow preventing necrotic area haemorrhage
(D) Extensive deposition of systemic amyloid within the affected organ

2013
159. Sludged blood condition is often reversible. It is most heavily dependent on:
(A) The absolute count of circulating white blood cells
(B) The ratio of plasma globulins and fibrinogen to albumin
(C) The mean arterial blood pressure within main vessels
(D) The exact concentration of glucose in the circulation

2004
160. Which statement INCORRECTLY describes a feature of cardiogenic shock?
(A) It results from a primary failure of the myocardial pumping action
(B) It can be precipitated by a massive acute myocardial infarction
(C) It can develop secondary to severe mechanical cardiac tamponade
(D) It features high cardiac output and massive systemic vasodilation

2006
161. Hydropic degeneration is most prominently observed in organs heavily dependent on ATP-driven ion pumps, such as the:
(A) Liver and kidneys
(B) Tendons and ligaments
(C) Cartilage
(D) Epidermis

2025
162. The pathological finding of "hyaline change" is most accurately considered a:
(A) Specific chemical entity with a defined molecular structure
(B) Specialized type of collagen located in basement membranes
(C) Descriptive, morphologic term for glassy, eosinophilic material
(D) Direct intracellular precursor leading to tissue calcification

2009
163. The beta-pleated sheet structure of amyloid is responsible for its unique staining characteristic, which is:
(A) Apple-green birefringence under polarized light with Congo red
(B) Metachromatic purple-red staining when using Toluidine blue
(C) Intense blue collagenous coloration with Masson's Trichrome
(D) Pronounced black lipid-positive staining with Sudan Black B

2025
164. In feline pathology, a condition termed "fatty liver" (hepatic lipidosis) is classically precipitated by:
(A) Excessive ingestion of high-protein dietary formulations
(B) Acute viral infection localized to the biliary tract ducts
(C) Chronic systemic iron overload leading to hemochromatosis
(D) Prolonged anorexia triggering massive lipid mobilization

2025
165. Glycogen accumulation in tissues can be confirmed histologically by utilizing the Periodic Acid-Schiff (PAS) stain alongside which diagnostic step?
(A) Acid-fast staining protocols to enhance structural contrast
(B) Diastase digestion to enzymatically remove the glycogen
(C) Congo red staining to demonstrate green birefringence
(D) Sudan III staining techniques to co-localize neutral lipids

2001
166. Ceroid is an endogenous pigment that differs from lipofuscin primarily because it:
(A) Does not contain lipid components in its biochemical structure
(B) Contains massive amounts of iron complexes within its matrix
(C) Is associated with pathological states rather than simple aging
(D) Is synthesized directly from tyrosine amino acid precursors

2013
167. Jaundice becomes clinically evident when serum bilirubin levels exceed:
(A) 0.1 mg/dL
(B) 0.5 mg/dL
(C) 2.0 to 2.5 mg/dL
(D) 10.0 mg/dL

2001
168. Which of the following conditions is an example of a pneumoconiosis?
(A) Silicosis
(B) Pulmonary tuberculosis
(C) Emphysema
(D) Bronchopneumonia

Click Here to Buy book on Amazon

2018

169. Metastatic calcification is typically seen in the context of:

(A) Normal serum calcium levels combined with local tissue necrosis
(B) Hypercalcaemia secondary to hyperparathyroidism or bone damage
(C) Hypocalcaemia associated with neuromuscular irritability and tetany
(D) Ischaemic infarction occurring within the functional myocardium

2019

170. Match the necrosis type with its distinct histopathological feature:

List I: (Necrosis Type)

P. Fibrinoid necrosis
Q. Fat necrosis
R. Liquefactive necrosis
S. Coagulative necrosis

List II: (Histopathological Feature)

1. Transformation of tissue into a viscous, fluid mass
2. Saponification showing chalky white deposits
3. Intense eosinophilic deposits in vessel walls
4. Preservation of cell outlines with loss of nuclei

(A) P-3, Q-2, R-1, S-4
(B) P-2, Q-3, R-1, S-4
(C) P-4, Q-2, R-1, S-3
(D) P-3, Q-1, R-2, S-4

2019

171. Dry gangrene is pathophysiologically a form of:

(A) Coagulative necrosis with desiccation
(B) Liquefactive necrosis modified by bacterial putrefaction
(C) Caseous necrosis resulting from mycobacterial infection
(D) Fibrinoid necrosis due to vasculitis

2019

172. During the early stages of metabolic stress, the dissociation of ribosomes from the rough endoplasmic reticulum represents:

(A) An irreversible step resulting in immediate, definitive cell death
(B) The onset of executioner caspase activation during active apoptosis
(C) A reversible shift to decrease protein synthesis and conserve energy
(D) The final step in safe, mature cytoplasmic autophagosome vesicle formation

2008

173. The primary reason a cell cannot survive lysosomal membrane rupture is that:

(A) Lysosomes synthesize the majority of cellular adenosine triphosphate
(B) The physical rupture forces mechanical tears in the plasma membrane
(C) It immediately triggers death-receptor extrinsic apoptotic pathway
(D) Released lysosomal enzymes digest essential cellular macromolecules

2019

174. Which event defines the onset of irreversible ischemic injury in the myocardium?

(A) Complete and rapid depletion of cytoplasmic glycogen stores
(B) Reversible cellular swelling due to sodium pump dysfunction
(C) Massive calcium influx causing permanent mitochondrial damage
(D) Severe progressive decline in intracellular physiological pH

2001

175. The accumulation of transudate in the peritoneal cavity is clinically termed:

(A) Hydrothorax
(B) Ascites
(C) Anasarca
(D) Hydropericardium

NODIA

2024

176. Which condition represents chronic passive congestion of the lungs?

(A) Active pulmonary hyperaemia occurring during intense aerobic exercise
(B) Right-sided heart failure leading to systemic passive hepatic congestion
(C) Local venous obstruction caused by a slow-growing neoplasm in the leg
(D) Left-sided heart failure leading to pulmonary capillary engorgement

2025

177. Ischaemia differs from hypoxia in that ischaemia also involves:

(A) Impaired removal of metabolic waste products such as lactic acid
(B) An increased delivery of oxygen to metabolically active tissues
(C) Significantly enhanced collateral blood flow to ischemic regions
(D) Immediate cell necrosis occurring without preceding reversible steps

2013

178. A haematoma is best defined as:
(A) A tiny punctate haemorrhage occurring on mucosal surfaces
(B) A systemic bleeding diathesis affecting clotting cascades
(C) A localized collection of blood outside the blood vessels
(D) Bleeding localized exclusively into synovial joint spaces

2011

179. A mural thrombus is defined as a thrombus that:
(A) Is attached to the cardiac wall or large arteries without fully occluding the lumen
(B) Completely occludes the microvascular lumen of small muscular veins and venules
(C) Forms exclusively on the surface of heart valves during infectious endocarditis
(D) Floats freely within the systemic arterial circulation as a thromboembolic fragment

2018

180. Identify the correct option regarding the Assertion (A) and Reason (R) given below:
Assertion (A): Irreversible cell injury is ultimately defined by the inability to reverse mitochondrial dysfunction and profound disturbances in membrane function.
Reason (R): Membrane damage allows massive calcium influx, which activates cellular enzymes that indiscriminately degrade structural proteins, DNA, and RNA.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2025

181. A paradoxical embolus requires the presence of which anatomical abnormality to occur?
(A) Advanced ulcerated atherosclerosis within the ascending aorta
(B) An arteriovenous shunt or patent interatrial septal defect
(C) Thrombophlebitis localized within deep veins of the pelvis
(D) A primary congenital aneurysm within pulmonary vasculature

2021

182. Red infarcts are most commonly found in the:
(A) Heart and kidneys
(B) Brain and spinal cord
(C) Lungs and intestines
(D) Spleen and liver

2022

183. Which statement accurately describes the clinical consequence of sludged blood?
(A) It drastically improves physiological tissue oxygenation
(B) It prevents development of systemic deep vein thrombosis
(C) It causes an immediate drop in systemic blood viscosity
(D) It leads to micro-infarctions and severe tissue ischaemia

2006

184. In the pathogenesis of septic shock, which microbial component is the most potent trigger of the severe systemic inflammatory response?
(A) Lipopolysaccharide endotoxin
(B) Peptidoglycan cell wall components
(C) Flagellar flagellin protein structures
(D) Soluble bacterial exotoxin molecules

2002

185. During cellular swelling, visually identifiable "clear vacuoles" in the cytoplasm primarily represent:
(A) Large lipid droplets accumulating in the cytoplasm
(B) Distended segments of the endoplasmic reticulum
(C) Phagolysosomes filled with digested cell debris
(D) Large accumulations of intracellular glycogen

2022

186. Which cellular alteration is considered a "protein folding defect" leading to specific toxic accumulations?
(A) Hyaline change within arteriolar vascular walls
(B) Hepatocellular alpha-1 antitrypsin deficiency
(C) Type I systemic glycogen storage disease state
(D) Acute sodium urate deposition in joint spaces

2018

187. The term "primary amyloidosis" refers to amyloidosis that is:
(A) Associated with plasma cell dyscrasias producing AL amyloid
(B) Associated with chronic infectious and inflammatory disease states
(C) Associated with genetic mutations in the

Downloaded from www.pdf.tube

transthyretin gene
(D) Associated with localized deposition of amyloid in brain tissue

2017
188. In contrast to fatty change (steatosis), "fatty infiltration" refers to:
(A) Abnormal triglyceride accumulation within parenchymal cells
(B) Abnormal lipid accumulation within systemic macrophages
(C) Accumulation of adipocytes in interstitial connective tissue
(D) Pathological cholesterol crystal deposition within arteries

2001
189. The presence of Armanni-Ebstein lesions in the kidneys is pathognomonic for:
(A) Severe urate nephropathy involving the renal interstitium
(B) Secondary systemic amyloidosis of the glomerular basement
(C) Hyaline droplet degeneration within proximal tubule cells
(D) Diabetic nephropathy inducing tubular glycogen accumulation

2008
190. Consider the following statements about gangrene:
I. Dry gangrene frequently affects internal organs like the intestine.
II. Moist gangrene is strictly a non-infectious process.
III. Gas gangrene involves crepitus produced by Clostridium spp.
IV. Dry gangrene presents with a clear line of demarcation.
V. Gangrene never involves coagulative necrosis.
Which of the above statements is/are INCORRECT?
(A) I, II, III and V
(B) I, II and V
(C) II, III and IV
(D) I, IV and V

2015
191. Bilirubin is the final breakdown product of:
(A) The globin portion of hemoglobin
(B) The porphyrin ring of heme
(C) The iron core of heme
(D) Myoglobin exclusively

2023
192. Which cells are primarily responsible for the fibrogenic response observed in chronic silicosis?
(A) Activated neutrophils releasing oxygen free radicals into tissue
(B) Eosinophils releasing tissue-destructive major basic protein
(C) Cytotoxic T-lymphocytes directly killing alveolar epithelial cells
(D) Alveolar macrophages secreting pro-fibrogenic cytokine molecules

2005
193. Which of the following is a characteristic feature of dystrophic calcification?
(A) Normal systemic calcium levels and localized cellular damage or necrosis
(B) Elevated systemic calcium levels throughout the circulation
(C) Excessive endocrine levels of circulating parathyroid hormone
(D) Completely intact and functional cellular architecture structures

NODIA

2002
194. Apoptosis differs fundamentally from necrosis in that apoptosis:
(A) Provokes a marked inflammatory response
(B) Always involves large contiguous tracts of tissue
(C) Involves cell shrinkage rather than cell swelling
(D) Results from catastrophic depletion of ATP

2011
195. Which of the following is a classic microscopic feature of liquefactive necrosis in the brain?
(A) Presence of lipid-laden gitter cells and cystic tissue spaces
(B) Presence of dead ghost cells with preserved structural outlines
(C) Presence of structured granulomas with epithelioid macrophages
(D) Presence of amorphous, friable, cheese-like caseous material

2008
196. Gas gangrene is characterized by extreme tissue destruction due to the release of:
(A) Systemic endotoxin released by Escherichia coli organisms
(B) Watery secretory cholera toxin released by Vibrio cholerae
(C) Neurotoxic tetanospasmin released by Clostridium tetani
(D) Alpha toxin lecithinase released by

Clostridium perfringens

2012
197. A key ultrastructural hallmark that a cell has transitioned into an irreversible injury state is:
(A) Progressive dilation of the rough endoplasmic reticulum
(B) Loss of cristae and severe mitochondrial membrane disruption
(C) Pronounced clumping of nuclear chromatin along the envelope
(D) Slight transient swelling of the cellular plasma membrane

2004
198. Cytoskeletal damage during cell injury is primarily mediated by the abnormal activation of:
(A) Active executioner caspases during apoptosis
(B) Nuclear RNA polymerases during transcription
(C) Calcium-dependent proteases like calpains
(D) Translating polyribosomes during synthesis

2017
199. Anasarca is defined as:
(A) Severe fluid accumulation confined entirely within the pleural space
(B) Localized subcutaneous oedema affecting a single distal extremity
(C) Oedema of the optic disc associated with high intracranial pressure
(D) Severe, generalized massive oedema with profound tissue swelling

2001
200. Consider the following statements regarding lipid metabolism disturbances:
I. Steatosis represents an absolute increase in intracellular lipids.
II. Fatty change is invariably irreversible and directly leads to cell death.
III. Alcohol abuse is a primary etiology for hepatic steatosis.
IV. Hypoxia can induce fatty change by impairing mitochondrial fatty acid oxidation.
V. Niemann-Pick disease is a sphingolipidosis.
Which of the above statements is/are correct?
(A) I, III, IV and V
(B) I, II and III
(C) II, III, IV and V
(D) III, IV and V

2005
201. Which organ is classically described as having "nutmeg" pathology in right-sided heart failure?
(A) Lung
(B) Liver
(C) Kidney
(D) Spleen

2016
202. The presence of haemosiderin-laden macrophages in the pulmonary alveoli indicates:
(A) Active pulmonary hyperaemia during acute exercise
(B) Chronic passive congestion of pulmonary tissues
(C) Acute lobar pneumonia with purulent exudation
(D) Pulmonary fat embolism following severe trauma

2013
203. Which mechanism of hypoxia is characterized by a failure of the cells to utilize delivered oxygen, as seen in cyanide poisoning?
(A) Histotoxic hypoxia
(B) Ischaemic hypoxia
(C) Hypoxaemic hypoxia
(D) Anaemic hypoxia

2007
204. Primary haemostasis culminates in the formation of:
(A) A stable fibrin mesh
(B) A mature red thrombus
(C) A primary platelet plug
(D) Plasminogen activation

2008
205. The primary anti-coagulant effect of intact endothelium involves the synthesis of:
(A) Procoagulant thromboxane A_2
(B) Procoagulant tissue factor
(C) Soluble blood fibrinogen
(D) Prostacyclin and nitric oxide

2023
206. Which type of embolism is most characteristically associated with fractures of long bones or severe trauma to adipose tissue?
(A) Air embolism
(B) Gas embolism
(C) Fat embolism
(D) Amniotic fluid embolism

2010
207. A characteristic morphological feature of an old, healed infarct in a solid organ like the kidney or spleen is a:
(A) Cystic cavity

Click Here to Buy book on Flipkart

(B) Depressed, pale fibrotic scar
(C) Hemorrhagic wedge
(D) Large purulent abscess

2024

208. Sludged blood reduces tissue perfusion primarily by increasing:
(A) Vascular dilation of major systemic arteries
(B) Total circulating vascular plasma volume
(C) Endothelial permeability across capillaries
(D) Blood viscosity in the microcirculation

2016

209. In the progressive stage of shock, which pathophysiological event is most prominent?
(A) Widespread tissue hypoxia and metabolic lactic acidosis
(B) Tachycardia with maintenance of normal arterial pressure
(C) Failure of baroreceptor reflexes causing cardiac arrest
(D) Excessive diuresis caused by renal tubular dysfunction

2023

210. Reversible cell injury is most fundamentally characterized by the loss of:
(A) Intracellular sodium loss and gain of intracellular potassium
(B) All intracellular organelle structures through karyolysis
(C) Intracellular potassium loss and gain of sodium and water
(D) The nuclear envelope integrity during enzymatic digestion

****

Downloaded from www.pdf.tube

ANSWERS

1. (D) Plasma colloid osmotic pressure
At the arterial end of capillary beds, fluid movement is governed by Starling forces. Capillary hydrostatic pressure drives fluid filtration out of the vasculature into the interstitial space. Conversely, plasma colloid osmotic pressure, which is exerted primarily by dissolved proteins such as albumin, pulls water back into the blood vessel, thereby opposing filtration and maintaining intravascular volume.

2. (C) High protein content with significant cellular debris
Inflammatory processes increase microvascular permeability, allowing large plasma proteins and cellular components to escape into the interstitial tissue, creating an exudate. This fluid features a high specific gravity and elevated protein concentration, alongside cellular debris. In contrast, non-inflammatory transudates result from hydrostatic or osmotic imbalances, yielding low protein content and minimal cellular presence.

3. (B) Dilation of central veins and sinusoids
Chronic passive congestion of the liver is initiated by systemic venous hypertension, which impedes outflow from the hepatic veins. This pressure backflow first induces dilation of the central veins and hepatic sinusoids. Subsequently, the prolonged hypoxia and hypoperfusion in these congested zones lead to centrilobular hepatocyte atrophy, fatty change, and necrosis, producing the characteristic nutmeg appearance.

4. (D) Mitochondrial oxidative phosphorylation
Ischaemia restricts blood supply, depriving cells of oxygen and essential substrates needed for aerobic metabolism. Without oxygen, the mitochondrial electron transport chain ceases to function, halting mitochondrial oxidative phosphorylation. This leads to a rapid drop in adenosine triphosphate production, impairing cellular membrane pumps, causing influx of sodium and water, and initiating cellular swelling and dysfunction.

5. (A) Haemorrhages measuring 1 to 2 cm in diameter
Ecchymoses are subcutaneous haemorrhages measuring approximately one to two centimeters in size. They are commonly referred to as bruises and represent larger accumulations of blood under the skin surface than petechiae or purpura. Over time, the extravasated red blood cells are degraded, leading to color changes from blue-red to green-yellow due to enzymatic breakdown of hemoglobin.

6. (C) Increased laminar blood flow rate
Virchow's triad outlines the three primary pathophysiological categories that contribute to thrombus formation. These factors consist of endothelial injury, hypercoagulability, and alterations in normal blood flow, specifically turbulence or stasis. Rapid laminar blood flow represents normal, healthy circulation that prevents platelets from contacting the endothelium, thereby actively discouraging thrombosis rather than predisposing a patient to clot formation.

NODIA

7. (A) Severe fracture of long bones
Fat embolism syndrome typically arises after traumatic injury to tissues containing significant adipose reserves or red bone marrow. Severe fractures of long bones, such as the femur or tibia, release microscopic fat globules from the marrow cavity into ruptured venules. These circulating lipids then migrate to the pulmonary and systemic microvasculature, causing mechanical obstruction and chemical pneumonitis.

8. (D) Tissues featuring dual blood supplies
Red or haemorrhagic infarcts occur primarily in tissues and organs with dual arterial supplies, such as the lungs or liver, and in loose tissues that permit blood accumulation. When blood flow is obstructed in one supply, the patent secondary vessel continues to deliver blood into the necrotic zone, resulting in extensive haemorrhage into the dead parenchymal tissue.

9. (B) Plasma protein changes causing rouleaux
The phenomenon of sludged blood occurs when changes in circulating plasma proteins alter the

surface charges of red blood cells. Specifically, elevations in large asymmetrical molecules like fibrinogen or globulins reduce the negative electrostatic forces between erythrocytes. This alteration promotes their clustering into stacks, known as rouleaux, which increases blood viscosity and slows capillary blood flow.

10. (A) P-2, Q-3, R-4, S-1
Different forms of shock arise from distinct pathophysiological mechanisms. Cardiogenic shock is caused directly by myocardial pump failure. Hypovolaemic shock results from critical loss of intravascular fluid volume. Septic shock is initiated by microbial endotoxins causing peripheral pooling and vasodilation. Anaphylactic shock is mediated by an IgE-driven hypersensitivity reaction, triggering systemic vasodilation and increased vascular permeability.

11. (D) Sodium-potassium ATPase pump
In reversible cellular injury, depleted intracellular adenosine triphosphate impairs the activity of the ATP-dependent sodium-potassium membrane pump. Consequently, sodium ions accumulate inside the cytoplasm while potassium ions diffuse out. This cellular solute imbalance creates an osmotic gradient that draws water into the cytosol, causing swelling of the organelles and the cell itself.

12. (A) A descriptive term for glassy, pink-staining material
Hyaline change is not a specific, single chemical entity or distinct disease process. Instead, it serves as a descriptive histological term denoting an intracellular or extracellular accumulation of various substances that appear homogeneous, glassy, and pink when stained with hematoxylin and eosin. It represents diverse pathological processes, including protein accumulation, amyloidosis, or chronic fibrosis.

13. (B) Congo Red
Amyloid is an abnormal extracellular proteinaceous deposit characterized by a beta-pleated sheet conformation. In histopathology, Congo red is the standard diagnostic stain used for its identification. When viewed under polarized light, Congo red-stained amyloid deposits exhibit a highly specific apple-green birefringence. This optical phenomenon uniquely distinguishes amyloid from other hyaline and extracellular protein deposits.

14. (B) Niemann-Pick disease is caused by an excess of circulating low-density lipoproteins.
Niemann-Pick disease is an inherited lysosomal storage disorder, not a condition caused by excess circulating low-density lipoproteins. It arises from genetic mutations causing a deficiency of the lysosomal enzyme acid sphingomyelinase. This enzymatic defect leads to the progressive accumulation of sphingomyelin within the lysosomes of phagocytic cells, particularly in liver, spleen, and brain tissues.

15. (B) Vasoconstriction → Platelet adhesion → Platelet activation → Platelet aggregation
Primary haemostasis begins immediately following endothelial injury. The sequence starts with reflex vasoconstriction to reduce local blood flow. Exposed subendothelial collagen then facilitates platelet adhesion through interaction with von Willebrand factor. Adhered platelets undergo activation, changing shape and releasing chemical mediators. These signals recruit additional platelets, driving platelet aggregation to form the temporary haemostatic plug.

16. (C) Previous free radical injury and aging
Lipofuscin is an insoluble brownish-yellow cytoplasmic pigment known as the wear-and-tear pigment. It is composed of polymers of lipids and proteins derived from the free radical-mediated peroxidation of subcellular membranes. Its accumulation in highly metabolic, non-dividing parenchymal cells, such as myocardial cells and neurons, is a classic hallmark of cellular aging and chronic atrophy.

17. (D) Unconjugated bilirubin
Pre-hepatic icterus is driven by excessive destruction of erythrocytes, which releases large quantities of hemoglobin into the blood. Macrophages break down this hemoglobin, producing substantial amounts of unconjugated bilirubin. This overproduction overwhelms the capacity of hepatocytes to conjugate the pigment with glucuronic acid, resulting in elevated serum levels of lipid-soluble unconjugated bilirubin.

18. (C) Carbon particles
Anthracosis is a common environmental pulmonary change caused by the inhalation of fine carbon or coal dust. Once inhaled into the deep alveolar spaces, these inert carbon particles are phagocytosed by alveolar macrophages. These pigment-laden macrophages then transport the

Click Here to Buy book on Amazon

carbon into the pulmonary interstitial tissue and regional lymph nodes, yielding a characteristic black discoloration.

19. (B) Normocalcaemia and areas of tissue necrosis
Dystrophic calcification occurs locally in degenerating or necrotic tissues, despite normal systemic calcium metabolism and normal serum calcium levels. Calcium ions accumulate on membrane-bound vesicles in damaged cells, serving as initiation sites for the crystallization of calcium phosphate. This is distinct from metastatic calcification, which occurs in viable tissues due to systemic hypercalcaemia.

20. (A) Both A and R are true and R is the correct explanation of A.
Apoptosis is a regulated form of cell death that does not elicit inflammation because the outer cell membrane remains structurally intact. Instead of leaking intracellular enzymes, the shrinking cells break down into membrane-bound apoptotic bodies. These structures express phosphatidylserine on their outer surface, signaling adjacent phagocytes to engulf them quietly before any inflammatory pathways are triggered.

21. (A) Liquefactive necrosis
In the central nervous system, hypoxic cell injury uniquely results in liquefactive necrosis rather than coagulative necrosis. Brain tissue contains high lipid and water content and lacks a dense connective tissue framework. Microglial cells and migrating neutrophils rapidly release hydrolytic enzymes that completely digest the dead neural parenchyma, transforming the infarcted area into a liquid viscous mass.

22. (C) It features a clear line of demarcation
Dry gangrene is caused primarily by slow arterial occlusion in extremities, leaving venous drainage intact. This allows the affected tissues to dehydrate and mummify, appearing dry, shrunken, and black. A clear inflammatory line of demarcation develops between the viable and dead tissue. Conversely, moist gangrene involves bacterial invasion, lack of demarcation, and rapid putrefaction.

23. (A) Mitochondrial swelling and clearing of the matrix
The earliest ultrastructural change in mitochondria during cellular stress is mitochondrial swelling accompanied by clearing of the inner matrix. This occurs because compromised ATP synthesis impairs ion regulation, leading to solute accumulation and water influx. If the stress is relieved, these changes revert, whereas severe membrane rupture or flocculent densities indicate transition to irreversible cell death.

24. (D) Lysosomal rupture and enzyme leakage
While autophagosome formation or mild matrix swelling can occur during reversible phases, lysosomal rupture and leakage of acid hydrolases into the cytosol represents a definitive hallmark of irreversible cell injury. The released enzymes, activated by the acidic intracellular environment, digest cytoplasmic and nuclear components, leading to enzymatic dissolution and cell death.

25. (B) Colloid osmotic pressure exceeds hydrostatic pressure.
At the venular end of the capillary bed, the hydrostatic pressure within the vessel decreases significantly due to resistance and fluid loss at the arterial end. Meanwhile, the concentration of non-diffusible plasma proteins remains elevated, keeping the colloid osmotic pressure high. Because this osmotic pressure exceeds the hydrostatic pressure, water is drawn back into the vasculature.

26. (C) Pulmonary oedema
Pulmonary oedema involves the rapid accumulation of fluid in the alveolar spaces and interstitial septa of the lungs. This physical barrier of fluid significantly increases the physical distance for gas exchange. Consequently, it severely hinders oxygen diffusion across the alveolar-capillary membrane into the bloodstream, leading to acute hypoxemia, respiratory failure, and death.

27. (D) Haemosiderin-laden macrophages
During chronic passive congestion of the lungs, elevated pulmonary venous pressure leads to the leakage of erythrocytes from capillaries into the alveolar spaces. Alveolar macrophages phagocytose these extravasated red blood cells and degrade their hemoglobin. This process generates intracellular haemosiderin pigment, transforming these cells into characteristic haemosiderin-laden macrophages, commonly referred to as heart failure cells.

NODIA

28. (A) Arterial thrombosis or embolism
Regional tissue ischaemia represents a localized reduction in blood perfusion to a specific tissue or organ. While systemic alterations like hypotension affect the whole body, localized blockages are most frequently caused by mechanical vascular occlusion. This occurs primarily due to either in-situ arterial thrombosis or the lodging of a circulating embolus in the narrow arterial lumen.

29. (C) 3 to 10 mm
Purpura is a specific classification of haemorrhage within the skin, mucous membranes, or serosal surfaces. These lesions are intermediate in size, measuring between three and ten millimeters in diameter. They are larger than pinpoint petechiae, which measure less than three millimeters, but smaller than ecchymoses, which typically exceed one to two centimeters in diameter.

30. (A) II and III
The coagulation cascade involves highly regulated enzymatic steps. The extrinsic pathway is initiated by tissue factor, whereas the intrinsic pathway begins with Factor XII. Both pathways converge at the activation of Factor X, representing a central step. Subsequently, thrombin, generated from prothrombin, converts soluble fibrinogen into insoluble fibrin monomers to stabilize the primary clot.

31. (A) Left heart chambers
Systemic thromboemboli travel through the arterial circulation and cause tissue infarction downstream. The vast majority of these emboli, approximately eighty percent, originate from mural thrombi located within the left chambers of the heart. These clots are often associated with myocardial infarction, atrial fibrillation, or valvular diseases, whereas venous thrombi typically embolize to the pulmonary circulation.

32. (C) Solid organs with end-arterial circulation like the kidney
White or anaemic infarcts typically occur in solid, parenchymal organs with end-arterial circulations, such as the kidney, spleen, or heart. Because these organs lack a dual blood supply or extensive anastomoses, arterial occlusion completely halts inflow to the downstream tissue. The dense nature of these solid organs further limits the seepage of blood from adjacent patent capillaries.

33. (D) Decreased microvascular tissue perfusion
The aggregation of erythrocytes in sludged blood markedly increases the overall viscosity of blood within the microcirculation. This altered physical state compromises capillary flow velocity and enhances resistance. Consequently, the primary physiological result is a significant decrease in microvascular tissue perfusion, which starves parenchymal cells of essential oxygen and vital nutrients, exacerbating ischemic injury.

34. (C) Activation of the sympathetic nervous system
In the initial compensatory stage of hypovolaemic shock, reduced blood volume triggers baroreceptors, prompting robust activation of the sympathetic nervous system. This autonomic response releases catecholamines, inducing peripheral vasoconstriction, increasing heart rate, and boosting myocardial contractility. These physiological mechanisms help maintain perfusion pressure to vital organs, temporarily offsetting the effects of volume depletion.

35. (B) Hydropic cellular swelling
Ballooning degeneration represents an extreme, severe form of hydropic cellular swelling. In viral infections such as poxviruses, cellular metabolism is disrupted, compromising membrane integrity and energy production. The failure of sodium-potassium pumps leads to a massive accumulation of intracellular water. Affected cells swell dramatically, appearing rounded, pale, and enlarged under light microscopy.

36. (D) Gout
Gout is a metabolic disease characterized by chronic hyperuricaemia, which leads to the precipitation of monosodium urate crystals. These needle-shaped crystals deposit preferentially in articular cartilage, synovial membranes, and surrounding periarticular soft tissues. This deposition triggers a severe, acute inflammatory response and can progress to form chronic, destructive inflammatory lesions known as tophi.

37. (A) AA amyloid
AA amyloid is the specific biochemical form deposited in secondary or reactive systemic amyloidosis, which complicates chronic inflammatory diseases such as rheumatoid arthritis or osteomyelitis. The liver synthesizes serum amyloid A, an acute-phase reactant, in

Downloaded from www.pdf.tube

response to inflammatory cytokines. Persistent elevation of this precursor leads to incomplete proteolysis, forming insoluble AA amyloid fibrils.

38. (C) Increased synthesis of triglycerides due to excess NADH
Alcohol metabolism in the liver by alcohol dehydrogenase and aldehyde dehydrogenase utilizes nicotinamide adenine dinucleotide, generating an excess of cellular NADH. This high NADH to NAD ratio inhibits mitochondrial fatty acid oxidation and promotes the synthesis of glycerol-3-phosphate. Consequently, there is an increased synthesis of triglycerides, leading to lipid accumulation within hepatocytes.

39. (A) Von Gierke disease
Von Gierke disease, or type I glycogen storage disease, is an autosomal recessive genetic disorder caused by a deficiency of the enzyme glucose-6-phosphatase. This enzymatic defect prevents the final step of glycogenolysis and gluconeogenesis. Consequently, large quantities of structurally normal glycogen accumulate pathologically within the cytoplasm of hepatocytes and renal proximal tubular epithelial cells.

40. (B) I and III
Melanin is an endogenous pigment synthesized by melanocytes through tyrosine oxidation. In contrast, lipofuscin is not an indicator of acute cellular injury but rather a wear-and-tear pigment denoting chronic aging or atrophy. Ceroid is an autofluorescent, lipofuscin-like pigment found in pathological conditions, and melanin is stored in melanosomes, not lysosomes of neutrophils.

41. (B) Represents iron overload without tissue injury
Haemosiderosis is a condition of systemic or localized iron overload characterized by the accumulation of haemosiderin within macrophages and parenchymal cells. Crucially, it does not cause associated parenchymal tissue damage or organ dysfunction. This contrast is key to haemochromatosis, a more severe, genetically determined disease where extensive iron deposition directly induces tissue injury, fibrosis, and organ failure.

42. (C) Alveolar macrophages
Inhaled silica particles reach the alveoli, where they are ingested by alveolar macrophages. Because silica is highly toxic and cannot be degraded, it disrupts lysosomal membranes within the macrophages, releasing enzymes that cause macrophage death. This process triggers a continuous cycle of recruitment, activation, and cell death, releasing fibrogenic cytokines that drive pulmonary fibrosis.

43. (D) Formation of mature bone in non-osseous soft tissues
Heterotopic ossification refers to the abnormal metaplastic formation of mature, lamellar bone within non-osseous soft tissues, such as skeletal muscle or connective tissue. This process typically occurs in response to trauma, surgery, or neurological injury, and involves the osteogenic differentiation of local mesenchymal progenitor cells rather than simple calcium salt precipitation.

44. (A) Caspases
The execution phase of apoptosis is mediated by a highly specialized family of aspartate-specific cysteine proteases known as caspases. Initiator caspases first activate executioner caspases, specifically caspase-3, -6, and -7. Once activated, these executioner proteases cleave vital structural and functional proteins, leading to the characteristic morphological alterations of programmed cell death.

NODIA

45. (A) II, IV, III, I
The extrinsic pathway of apoptosis begins when the extracellular ligand FasL binds to the transmembrane Fas receptor. This binding event initiates the intracellular recruitment of adaptor proteins, leading to the formation of the death-inducing signaling complex. Consequently, initiator Caspase-8 is recruited and activated, which then cleaves and activates executioner caspases to execute programmed cell death.

46. (B) Structureless, granular, cheese-like debris
Caseous necrosis is a distinct morphological form of cell death most commonly associated with tuberculous infections. Histologically, the necrotic focus consists of structureless, granular, eosinophilic debris containing amorphous lipids and proteins. Unlike coagulative necrosis, the original tissue architecture and cellular outlines are completely obliterated, and the necrotic area is typically enclosed within a granulomatous inflammatory border.

47. (A) Clostridium
Gas gangrene, a highly lethal form of moist gangrene, is caused by anaerobic, spore-forming bacteria belonging to the genus Clostridium, most notably Clostridium perfringens. These bacteria contaminate deep, traumatic wounds where they proliferate in anaerobic conditions, releasing necrotizing toxins and enzymes that destroy muscle tissues and ferment carbohydrates, producing characteristic gas bubbles in the tissue.

48. (C) Decreased cellular protein synthesis
During acute cellular stress, such as hypoxia or ATP depletion, structural changes occur within the endoplasmic reticulum. The physical detachment or dissociation of ribosomes from the membrane of the rough endoplasmic reticulum impairs translation. Without intact ribosomal-membrane interactions, the cell is unable to properly synthesize proteins, resulting in a marked decrease in total cellular protein synthesis.

49. (D) Massive calcium influx and enzyme leakage
Irreversible plasma membrane damage destroys the cell's selective permeability barrier. This disruption allows extracellular calcium ions, which exist at much higher concentrations outside the cell, to flood the cytosol. This massive calcium influx activates destructive intracellular enzymes, such as phospholipases and proteases, while cellular proteins and enzymes leak out into the extracellular space.

50. (B) P-3, Q-2, R-1, S-4
Pathological pigments possess distinct biological origins. Melanin is a tyrosine-derived endogenous pigment synthesized by melanocytes. Haemosiderin is an iron-containing storage aggregate representing catabolized hemoglobin. Lipofuscin is a wear-and-tear pigment composed of lipid-protein polymers from membrane peroxidation. Bilirubin is a porphyrin ring degradation product formed during the breakdown of heme in the mononuclear phagocyte system.

51. (B) Increased capillary hydrostatic pressure
In patients with severe right-sided heart failure, the weakened right ventricle is unable to pump blood forward effectively, causing blood to pool in the systemic venous system. This systemic venous congestion directly elevates hydrostatic pressure within the capillaries. This increased pressure forces excess fluid out of the microvasculature into the interstitium, producing a non-inflammatory transudate.

52. (D) Failure to drain interstitial fluid and proteins
Normally, lymphatic vessels play a critical role in returning the small amount of filtered interstitial fluid and extravasated proteins back into the venous circulation. When lymphatic drainage is obstructed, this fluid and its protein content accumulate in the interstitial spaces. This retention increases interstitial colloid osmotic pressure, further hindering fluid resorption and causing localized lymphoedema.

53. (A) Increased arterial inflow of blood to tissues
Active hyperaemia is a physiological process characterized by an increased volume of blood in a tissue, driven by arteriolar dilation. This dilation increases the inflow of arterial blood to the microvascular bed. It commonly occurs in response to increased metabolic activity, such as in skeletal muscle during exercise, or as an early response to inflammatory stimuli.

54. (C) Depletion of intracellular ATP
Hypoxia induced by ischaemia immediately restricts the oxygen required for mitochondrial oxidative phosphorylation. Consequently, the earliest cellular consequence is a rapid depletion of intracellular adenosine triphosphate. Because ATP is essential for cellular homeostasis, this loss impairs energy-dependent membrane transport systems, shifting cellular metabolism toward anaerobic glycolysis and initiating the cascade of cellular injury.

55. (A) Platelet dysfunction
Petechiae are minute, pinpoint haemorrhages measuring one to two millimeters in diameter that occur on skin or mucosal surfaces. They are most classically associated with quantitative or qualitative platelet defects, such as thrombocytopenia or platelet dysfunction, and local increases in intravascular pressure or vascular wall fragility, rather than coagulation factor deficiencies.

56. (D) Factor V Leiden mutation
Hypercoagulable states are divided into primary, genetic disorders and secondary, acquired conditions. Factor V Leiden mutation is the most common inherited cause of hypercoagulability.

Click Here to Buy book on Flipkart

This single nucleotide mutation renders Factor V resistant to cleavage and inactivation by activated protein C, leading to an unregulated, prothrombotic state that significantly increases the risk of deep vein thrombosis.

57. (B) Air (nitrogen) embolus
Decompression sickness, or Caisson disease, occurs when a rapid decrease in atmospheric pressure causes dissolved gases to come out of solution. Because nitrogen is highly soluble in body lipids and tissues at high pressure, a rapid ascent causes it to form physical bubbles in the blood and tissues, acting as gas emboli that obstruct microvessels.

58. (C) Necrosis, acute inflammation, scavenging, and granulation
Following tissue ischaemia, cells undergo ischemic necrosis, which releases cellular contents into the interstitium. This necrotic debris triggers an acute inflammatory response, recruiting neutrophils to the site. Over subsequent days, macrophages infiltrate the area to scavenge and phagocytose the dead cells, followed by the formation of highly vascularized granulation tissue that eventually matures into a fibrous scar.

59. (D) Impairing perfusion and increasing viscosity
Sludged blood is characterized by the physical clump of erythrocytes, which significantly raises local blood viscosity within the microvasculature. This increased viscosity slows capillary blood flow and elevates resistance, impairing adequate perfusion of downstream tissues. Consequently, the delivery of oxygen and nutrients to parenchymal cells is compromised, directly worsening local hypoxic cellular injury.

60. (A) Both A and R are true and R is the correct explanation of A.
Septic shock is characterized by systemic vasodilation and increased vascular permeability leading to tissue hypoperfusion. This life-threatening condition is initiated when bacterial endotoxins stimulate the host immune system, triggering a massive release of inflammatory cytokines, such as tumor necrosis factor and interleukin-1. These mediators induce widespread endothelial activation, leading to systemic hemodynamic collapse.

61. (B) Clear cytoplasmic vacuoles from distended ER
Hydropic degeneration is an early, reversible form of cellular injury characterized by the accumulation of water inside the cell. Under a light microscope, this fluid accumulation is visible as numerous small, clear vacuoles within the cytoplasm. These vacuoles represent the physical swelling and distension of the endoplasmic reticulum cisternae due to the failure of cellular volume control.

62. (B) Glycosaminoglycans
Mucoid or myxomatous degeneration is a connective tissue alteration characterized by the excessive accumulation of hydrophilic glycosaminoglycans and proteoglycans in the extracellular matrix. This overproduction disrupts the normal arrangement of collagen and elastic fibers, imparting a pale, basophilic, gelatinous appearance to the affected tissue under histological examination, commonly observed in mitral valve prolapse.

63. (A) Pressure atrophy of adjacent functional cells
Amyloid is an insoluble, fibrillar protein deposit that accumulates in the extracellular space of various organs. Because the body cannot easily degrade these beta-pleated sheet proteins, the progressive expansion of amyloid deposits physically compresses the adjacent parenchymal cells. This chronic mechanical compression deprives cells of blood supply and nutrients, leading to pressure atrophy and organ failure.

64. (C) Deficiency of specific lysosomal enzymes
Sphingolipidoses, including Tay-Sachs disease, are inherited metabolic disorders characterized by the pathological accumulation of specific lipids within cells. These diseases are caused by genetic mutations that lead to a deficiency of a specific lysosomal hydrolase enzyme. Without this enzyme, the targeted lipid substrate cannot be degraded, resulting in its progressive accumulation within lysosomes.

NODIA

65. (D) Epithelium of the renal proximal tubules
In patients with poorly controlled diabetes mellitus, severe hyperglycemia leads to high concentrations of glucose in the glomerular filtrate. This excess glucose exceeds the reabsorptive capacity of the kidneys and is actively taken up by the epithelial cells of the renal proximal tubules. Once inside these cells,

the glucose is synthesized into glycogen, causing massive intracellular accumulation.

66. (C) Lipofuscin
Lipofuscin is an insoluble, yellowish-brown cytoplasmic pigment that accumulates progressively in cells as they age or undergo chronic atrophy. Often termed the wear-and-tear pigment, it consists of complex aggregates of lipids and proteins generated through the free radical-driven peroxidation of lipids in subcellular membranes, remaining indefinitely stored within cytoplasmic lysosomal residual bodies.

67. (B) Heme
Porphyrias represent a heterogeneous group of rare inherited or acquired metabolic disorders. They are characterized by enzymatic defects in the biosynthetic pathway of heme, which is essential for hemoglobin, myoglobin, and cytochrome enzymes. This enzymatic blockade leads to the abnormal accumulation and excretion of porphyrins or their chemical precursors in various tissues.

68. (D) Silicosis
Silicosis is a chronic, occupational fibrotic lung disease caused by the inhalation of crystalline silica dust. Ingested silica particles kill alveolar macrophages, releasing fibrogenic cytokines that drive the formation of characteristic dense collagenous nodules in the lungs. This continuous macrophage destruction impairs cell-mediated intelligence, significantly increasing the patient's susceptibility to pulmonary tuberculosis.

69. (A) It requires the presence of hypercalcaemia.
Metastatic calcification involves the deposition of calcium salts in normal, viable tissues, which is driven primarily by systemic hypercalcaemia. This elevated calcium blood level can result from hyperparathyroidism, bone destruction, or vitamin D toxicity. In contrast, dystrophic calcification occurs locally within damaged or necrotic tissues, while systemic calcium levels remain completely normal.

70. (B) I and II
Necrosis is a form of cell death characterized by cell swelling, membrane rupture, and the leakage of intracellular contents, which inevitably incites an inflammatory response. Fibrinoid necrosis is characterized by immune complex deposition and fibrin leakage in blood vessel walls. Coagulative necrosis is typical of solid organs, whereas brain infarcts exhibit liquefactive necrosis.

71. (A) Pancreas
Fat necrosis is a specialized pattern of cell death occurring in adipose tissue. It is most classically caused by the pathological release of activated digestive lipases from the injured pancreas during acute pancreatitis. These enzymes hydrolyze fat cells into free fatty acids, which then combine with tissue calcium to form chalky white deposits through saponification.

72. (D) Rapid spread with putrefaction and toxemia
Moist gangrene represents a highly dangerous clinical emergency. It typically arises from venous congestion combined with bacterial infection of necrotic tissues. The presence of putrefactive bacteria causes the tissue to swell, soften, and decay rapidly. Because there is no distinct line of demarcation, bacterial toxins enter the systemic blood flow, leading to severe systemic toxemia and shock.

73. (B) The detachment of ribosomes
During early reversible cellular injury, depleted ATP levels cause cellular swelling. This osmotic imbalance leads to water accumulation within the endoplasmic reticulum, resulting in its physical dilation and swelling. As the endoplasmic reticulum cisternae expand, ribosomes detach from the rough outer membrane, halting protein translation and leading to a significant decrease in protein synthesis.

NODIA

74. (C) Breakdown of filaments leading to membrane fragmentation
In irreversible cell injury, massive calcium influx activates calcium-dependent intracellular proteases, such as calpains. These enzymes degrade key structural components of the cytoskeleton, including actin filaments and microtubules. This enzymatic breakdown deprives the plasma membrane of its structural support, leading to extensive membrane blebbing, cytoplasmic fragmentation, and eventual rupture of the cell.

75. (D) $J_v = K_f[(P_c - P_i) - (\pi_c - \pi_i)]$
The movement of fluid across a capillary wall is governed by Starling forces, which describe the balance between hydrostatic and osmotic pressures. The net fluid filtration rate depends on the capillary filtration coefficient multiplied

by the differences in hydrostatic pressures minus the differences in oncotic pressures between the capillary lumen and the interstitial space.

76. (A) Increased microvascular permeability
An exudate is an inflammatory fluid that accumulates in interstitial spaces. It is fundamentally caused by increased microvascular permeability, which is driven by inflammatory mediators like histamine or bradykinin that induce endothelial cell contraction. This allows plasma proteins, cellular elements, and fluids to escape into tissues, resulting in a protein-rich fluid with a high specific gravity.

77. (B) Centrilobular congestion and fatty change
The distinctive "nutmeg" gross appearance of the liver in chronic passive congestion arises from contrasting zones of cellular changes. Congestion and hypoxia are most severe in the centrilobular zones, producing dark reddish-brown areas of dilated central veins and sinusoids. Conversely, the less hypoxic periportal hepatocytes undergo reversible fatty change, appearing as pale, yellowish-brown zones.

78. (A) Loss of ATP → Na^+/K^+ pump failure → Cellular swelling
Ischaemia restricts oxygen supply, shutting down mitochondrial oxidative phosphorylation and causing a rapid loss of intracellular ATP. Lacking energy, the ATP-dependent sodium-potassium pumps in the plasma membrane fail to maintain ion gradients. Consequently, sodium ions accumulate inside the cell, creating an osmotic gradient that draws water into the cytosol, leading to hydropic cellular swelling.

79. (A) Thrombin
The final step of the coagulation cascade is crucial for stabilizing the primary platelet plug. Thrombin, a highly active serine protease generated from its precursor prothrombin, directly cleaves fibrinopeptides from soluble fibrinogen molecules. This enzymatic cleavage allows the remaining fibrin monomers to spontaneously polymerize into insoluble fibrin strands, which are then cross-linked.

80. (A) I and II
Red or haemorrhagic infarcts typically occur in loose tissues or organs with dual blood supplies, such as the lungs and small intestine, rather than solid organs. Conversely, white or anaemic infarcts occur in solid organs with end-arterial circulation, such as the kidney or spleen. Infarcted tissues generally undergo coagulative necrosis, with the exception of liquefactive necrosis in the brain.

81. (D) Strongylus vulgaris
In veterinary medicine, parasitic emboli are a well-documented cause of tissue ischemia and infarction. This condition is most classically associated with the migration of Strongylus vulgaris larvae in horses. The larval forms migrate into the intima of the cranial mesenteric artery, inducing local endarteritis and thrombosis. Fragments of these thrombi or larvae can embolize, causing colic.

82. (C) Occluded blood vessel
Infarcts in organs like the spleen or kidneys assume a characteristic wedge shape determined by vascular branching. The apex of this necrotic wedge points directly toward the site of the occluded blood vessel, while the wide base corresponds to the peripheral boundary of the tissue, which is often situated along the outer capsule of the organ.

83. (B) Reduced surface charge from plasma proteins
Erythrocytes naturally repel each other in circulation due to their negative surface charge, known as the zeta potential. In pathological states, elevations in positive, asymmetrical plasma proteins, such as fibrinogen or immunoglobulins, coat the red blood cells. This coating effectively reduces their negative surface charge, facilitating aggregation and clump formation within the microcirculation.

84. (D) Progressive stage
The progressive stage of shock represents a critical phase where compensatory mechanisms fail. As systemic tissue hypoperfusion persists, oxygen levels plummet, forcing cells to shift from aerobic respiration to anaerobic glycolysis. This metabolic alteration leads to excessive lactic acid production and systemic acidosis, which biochemically blunts arteriolar vasomotor responses, causing blood pooling and worsening hypoperfusion.

85. (A) The membrane is intact and injury can revert
Cellular swelling represents an early manifestation of cellular injury that remains fully reversible if

Downloaded from www.pdf.tube

the underlying noxious stimulus is removed. This reversibility is possible because the plasma and organellar membranes remain structurally intact, and critical enzymes have not leaked. Under these conditions, restoring energy production allows membrane pumps to function again, reclaiming volume homeostasis.

86. (C) Monosodium urate
Gout is a metabolic disorder resulting from persistent hyperuricaemia, which causes the accumulation and subsequent precipitation of uric acid. This process results in the deposition of needle-shaped monosodium urate crystals within articular cartilage, synovial membranes, and periarticular tissues. This crystalline accumulation triggers an inflammatory cascade, recruiting neutrophils that cause severe joint destruction.

87. (A) Serum amyloid-associated protein
In secondary systemic amyloidosis, the AA fibril deposits are derived from serum amyloid A, an acute-phase reactant synthesized primarily by hepatocytes. During chronic inflammation, elevated levels of inflammatory cytokines, including interleukin-1 and interleukin-6, stimulate the liver to overproduce this precursor, which undergoes incomplete proteolysis to form insoluble amyloid fibrils in tissues.

88. (D) Cytoplasmic lipid vacuoles displacing the nucleus
Steatosis, or fatty change of the liver, involves the abnormal accumulation of triglycerides within hepatocytes. Histopathologically, this manifests as clear, well-circumscribed lipid vacuoles in the cytoplasm. In macrovesicular steatosis, these growing vacuoles fuse to form a single large vesicle that pushes the hepatocyte cytoplasm and nucleus to the periphery of the cell.

89. (B) Inherited deficiencies of glycogen enzymes
Glycogen storage diseases, or glycogenoses, are a group of inherited metabolic disorders characterized by the abnormal accumulation of glycogen in tissues. They are caused by genetic mutations that lead to deficiencies of specific enzymes involved in either glycogen synthesis or degradation, preventing proper glycogen utilization and causing progressive damage to the liver, kidneys, or muscles.

NODIA

90. (A) P-3, Q-4, R-2, S-1
Different patterns of tissue necrosis correspond to specific clinical conditions. Coagulative necrosis is typical of myocardial infarction. Liquefactive necrosis is characteristic of cerebral infarction due to rapid enzymatic digestion. Caseous necrosis is most classically observed in granulomas during tuberculosis infections. Fat necrosis is associated with acute pancreatitis, where released lipases break down local adipose tissue.

91. (D) Obstruction of the biliary tree
Post-hepatic or obstructive icterus is caused by physical blockages in the biliary system downstream of the liver. When the common bile duct is obstructed, such as by a gallstone or tumor, conjugated bilirubin cannot flow into the intestine. This blockage forces the water-soluble conjugated bilirubin back into the hepatic sinusoids, elevating serum levels of conjugated bilirubin.

92. (A) Carbon dust
Inhaled carbon dust is the most common exogenous pigment encountered in pathology. Inhalation of coal dust or urban air pollution delivers these carbon particles deep into the alveolar spaces. There, alveolar macrophages phagocytose the pigment and transport it through lymphatic vessels to the pulmonary interstitium and hilar lymph nodes, producing characteristic black tissue discoloration.

93. (B) Atherosclerotic plaques in large arteries
Dystrophic calcification occurs locally in areas of chronic necrosis or lipid deposition, despite normal systemic calcium levels. A classic site for this process is within advanced atherosclerotic plaques in the walls of large arteries. Degenerating foam cells and extracellular lipid cores release membrane vesicles that serve as nucleating sites, driving the progressive crystallization of calcium phosphate.

94. (B) Are membrane-bound and do not leak contents
Apoptosis is a highly controlled form of programmed cell death. During this process, the cell shrinks and pinches off into multiple membrane-bound structures called apoptotic bodies. Because these vesicles possess intact plasma membranes, they contain and isolate cytoplasm and nuclear fragments without leaking

enzymes. Consequently, they are quietly engulfed by phagocytes without inducing inflammation.

95. (A) Blood vessels
Fibrinoid necrosis is a specialized pattern of tissue damage observed under light microscopy, primarily located within the walls of blood vessels. It classically arises from immune complex deposition, where antibody-antigen complexes accumulate in arterial walls. This deposition recruits inflammatory cells and triggers plasma protein leakage, which forms a bright pink, structureless band resembling fibrin.

96. (C) Crepitus from bacterial gas production
Both gas gangrene and moist gangrene feature rapid necrosis and bacterial invasion. However, gas gangrene is distinguished by the presence of crepitus during clinical palpation. Crepitus represents the physical sensation of bubbling under the skin, which is caused by the systemic release of gases fermented by anaerobic Clostridium species within deep muscle tissues.

97. (D) Whorls of lipids from damaged cell membranes
Ultrastructurally, myelin figures appear as large, laminated whorls of phospholipid membranes in the cytoplasm of damaged cells. They are generated from the breakdown and degradation of damaged organellar and cellular membranes during reversible or irreversible cell injury. These lipid residues are relatively resistant to immediate enzymatic degradation, aggregating into whorled structures resembling myelin.

98. (C) Severe mitochondrial and membrane dysfunction
Although many early cellular alterations are reversible, transition to irreversible injury represents a definitive tipping point. This point of no return is most closely linked to two critical events: severe mitochondrial dysfunction, preventing ATP regeneration, and profound loss of plasma and lysosomal membrane integrity, allowing unchecked calcium influx and the leakage of digestive enzymes.

99. (B) Albumin
Intravascular colloid osmotic pressure is the primary force drawing interstitial water back into capillary lumens. This pressure is exerted by large dissolved plasma proteins that cannot easily cross the endothelial barrier. Albumin is the most abundant protein in plasma, accounting for roughly seventy to eighty percent of this total osmotic pulling force.

100. (A) Both A and R are true and R is the correct explanation of A.
Ischaemia causes faster and more severe cellular injury than simple hypoxia. Hypoxia allows glycolysis to continue because glucose delivery remains intact, whereas ischaemia completely halts blood flow, stopping the delivery of substrates for anaerobic glycolysis and preventing the removal of toxic metabolic waste products, leading to rapid intracellular acidosis and injury.

101. (A) Impaired venous drainage from a tissue
Passive congestion is a pathological process characterized by an abnormal accumulation of venous blood in a tissue. It is caused by impaired venous drainage, which can arise from localized vascular blockages or systemic cardiac dysfunction, such as congestive heart failure. The blood accumulates in capillaries, leading to microvascular distension, hypoxic tissue injury, and local cyanosis.

102. (C) Eosinophilia and loss of cell detail
Under a light microscope, one of the earliest morphological signs of ischemic cellular injury is increased cytoplasmic eosinophilia, appearing brighter pink. This color change occurs because of the denaturation of cytoplasmic proteins and the loss of basophilic ribosomal RNA. Concurrently, the affected tissue exhibits a progressive loss of normal cellular detail and margins.

103. (A) Platelets and subendothelial collagen
During primary haemostasis, vascular injury exposes subendothelial collagen fibers to circulating blood. von Willebrand factor, synthesized by endothelial cells and megakaryocytes, binds directly to this exposed collagen matrix. Simultaneously, vWF binds to the glycoprotein Ib receptors on circulating platelets, acting as a crucial molecular bridge that mediates the initial step of platelet adhesion.

104. (D) Exposure of tissue factor and matrix
Under physiological conditions, healthy endothelium exerts anti-thrombotic properties. However, physical injury or activation of endothelial cells shifts their function to a pro-thrombotic state. The most direct pro-coagulant effect is the exposure of tissue factor

Click Here to Buy book on Amazon

and subendothelial matrix components, which initiates the extrinsic pathway of coagulation and recruits platelets to form a thrombus.

105. (A) Tissue Factor → Factor Xa → Thrombin → Fibr
The extrinsic coagulation cascade is initiated when vascular injury releases Tissue Factor, which binds and activates Factor VII. This complex directly activates Factor X to form Factor Xa. Factor Xa then cleaves prothrombin into active thrombin. Once generated, thrombin converts soluble fibrinogen into insoluble fibrin monomers, stabilizing the vascular clot.

106. (B) Originates in the venous system and enters systemic arteries via a cardiac defect
Paradoxical embolism occurs when a thrombus originating in the systemic venous circulation bypasses the lungs to enter systemic arteries. This abnormal passage typically happens through an interatrial or interventricular septal defect in the heart. Consequently, a deep vein thrombosis can travel directly to the brain, causing an ischemic stroke instead of a pulmonary embolism.

107. (A) Reperfusion of previously ischemic tissue
Hemorrhagic or red infarcts are highly likely to occur when blood flow is restored to a previously ischemic area. This process of reperfusion forces blood into the damaged, necrotic vascular bed, causing blood to leak extensively into the surrounding tissue. This characteristic leakage transforms what would be a pale infarct into a hemorrhagic one.

108. (C) Inducing capillary stasis and widespread microvascular thrombosis
Sludged blood refers to the aggregation of red blood cells into clumps within the microvasculature. This physical aggregation increases local blood viscosity, leading to capillary stasis and widespread microvascular thrombosis. This stasis blocks blood flow through small capillaries, depriving the surrounding tissue of vital oxygen and causing widespread ischemic cellular injury.

109. (D) IgE-mediated mast cell degranulation triggering systemic vasodilation
Anaphylactic shock is a systemic hypersensitivity reaction initiated by immunoglobulin E binding to mast cells. This interaction triggers rapid mast cell degranulation, releasing large amounts of histamine and other vasoactive mediators into the bloodstream. These mediators cause sudden, widespread vasodilation and increased capillary permeability, leading to severe hypotension and tissue hypoperfusion.

110. (C) I and III
Reversible cell injury is characterized by early structural changes, including cellular swelling and the formation of myelin figures from damaged membranes. While mitochondrial swelling occurs, severe mitochondrial vacuolization and outer membrane rupture are hallmarks of irreversible cell injury. Thus, only myelin figures and hydropic change represent the reversible stages of cellular damage.

111. (B) Abnormal intracellular accumulation of folded proteins
Russell bodies are round, eosinophilic inclusions that represent excessive accumulations of newly synthesized immunoglobulins within the rough endoplasmic reticulum of active plasma cells. This pathological process serves as a classic example of intracellular protein accumulation. It occurs when the rate of protein synthesis exceeds the cell's capacity to transport or secrete them.

112. (D) Non-branching beta-pleated sheet conformation
Amyloid proteins represent a diverse group of extracellular proteinaceous deposits that share a common physical structure. Regardless of their precursor origin, these proteins fold into unique, non-branching fibrils. These fibrils are arranged in a highly stable beta-pleated sheet conformation, which accounts for their resistance to normal physiological proteolysis and distinctive staining characteristics.

NODIA

113. (A) Foam cells
Macrophages that have phagocytosed excessive amounts of lipids, particularly cholesterol and its esters, accumulate these molecules within their cytoplasm. This intracellular lipid storage gives the cytoplasm a highly vacuolated, frothy appearance under the microscope. In histopathology, these lipid-laden macrophages are universally referred to as foam cells, which are key in atherosclerosis.

114. (C) Myofibers of the cardiac and skeletal muscle systems
Pompe disease is a lysosomal storage disorder caused by an inherited deficiency of the enzyme acid alpha-glucosidase. This deficiency prevents the breakdown of lysosomal glycogen, leading to its massive accumulation. The primary targets of this progressive glycogen accumulation are the myofibers of cardiac and skeletal muscles, resulting in severe cardiomegaly and weakness.

115. (A) Caspase 3 activation
Caspase three is a primary executioner protease in the apoptotic pathway, whose enzymatic activation marks an irreversible commitment to programmed cell death. Detecting this active enzyme serves as a definitive biochemical marker for apoptosis. This activation occurs in the early stages of the process, well before characteristic morphological changes are visible under light microscopy.

116. (D) Aggregates of ferritin micelles
Hemosiderin is an endogenous, iron-containing pigment that forms golden-yellow, granular aggregates within the cytoplasm. This pigment is primarily composed of large aggregates of ferritin micelles, which are complexes of ferric iron and apoferritin. It represents a localized or systemic excess of iron, where storage capacity is overwhelmed, causing ferritin to coalesce into visible granules.

117. (B) Malignant mesothelioma
Asbestos exposure is directly and specifically linked to the development of malignant mesothelioma, an aggressive neoplasm arising from the mesothelial lining of the pleura or peritoneum. Inhaled asbestos fibers persist in the lungs and migrate to mesothelial surfaces, where they induce chronic inflammation, cell damage, and neoplastic transformation over a long latency period.

118. (C) It is driven by the necrosis of underlying tissues.
Metastatic calcification is driven by systemic hypercalcemia or hyperphosphatemia, resulting in calcium deposition in normal, healthy tissues throughout the body. Unlike dystrophic calcification, which occurs locally in dying or dead tissues under normal calcium levels, metastatic calcification does not require tissue necrosis. Therefore, the presence of necrotic tissue is irrelevant to its pathogenesis.

119. (D) Internucleosomal DNA cleavage yielding fragments in multiples of ~200 base pairs
During the execution phase of apoptosis, specific calcium- and magnesium-dependent endonucleases are activated within the nucleus. These enzymes cleave genomic DNA at the vulnerable internucleosomal linker regions, sparing the nucleosome-associated segments. This targeted enzymatic action yields DNA fragments in precise multiples of approximately two hundred base pairs, producing a classic ladder on electrophoresis.

120. (A) I and II
Coagulative necrosis temporarily preserves the basic structural outline of dead cells and tissues for several days because denaturing conditions block proteolysis. Liquefactive necrosis is characterized by complete enzymatic digestion of tissue, commonly triggered by bacterial or fungal infections. These two forms are distinct, and their characteristic features are accurately described in these statements.

121. (B) The distal portions of the lower extremities
Dry gangrene is a clinical variant of coagulative necrosis that develops in distal extremities, particularly the toes and feet. It occurs when arterial blood supply is obstructed, causing tissue ischemia and subsequent desiccation. The affected tissue becomes dry, shriveled, and dark, presenting a sharp line of demarcation separating it from healthy tissue.

122. (B) Loss of mitochondrial matrix granules
In early metabolic stress, a decrease in the cellular energy state directly affects mitochondrial function. The loss of normal, electron-dense mitochondrial matrix granules, which store divalent cations like calcium, serves as a direct morphological indicator of depleted ATP production. This change reflects altered ionic homeostasis before irreversible, high-amplitude mitochondrial swelling occurs in the cell.

123. (A) Amorphous densities within the mitochondrial matrix
The transition from reversible to irreversible cell injury is marked ultrastructurally by the appearance of large, amorphous densities within the mitochondrial matrix. These densities

Downloaded from www.pdf.tube

represent precipitated proteins, lipids, and calcium, indicating severe, irreversible damage to mitochondrial membrane integrity and a complete loss of the cell's capacity to regenerate vital adenosine triphosphate molecules.

124. (C) Movement of fluid into the interstitium causing edema
According to Starling's law of capillary fluid dynamics, a decrease in plasma colloid osmotic pressure reduces the inward force retaining water within the vascular space. This disruption allows the outward hydrostatic pressure to dominate, forcing excess fluid to move from the capillary lumen into the surrounding interstitial compartment, resulting in clinical edema or effusion.

125. (D) Greater than 1.020
An exudate is an inflammatory fluid characterized by a high concentration of proteins, cellular debris, and inflammatory cells. Because of these abundant dissolved proteins and cellular components, the specific gravity of an exudate is elevated, typically exceeding one point zero two zero, which distinguishes it from a low-protein, low-cellular transudate in tissue spaces.

126. (C) Local release of nitric oxide and adenosine molecules
Active hyperemia in skeletal muscle during physical exercise is a physiological response driven by increased metabolic demand. The contracting muscle fibers and endothelial cells release local vasodilators, primarily nitric oxide and adenosine. These molecules relax the surrounding arteriolar smooth muscle, increasing local blood flow to match the metabolic needs of the active tissue.

127. (B) Neurons
Neurons are highly susceptible to hypoxic-ischemic injury due to their high metabolic rate and lack of significant glycogen reserves. Consequently, they depend entirely on a continuous supply of oxygen and glucose for ATP production. While fibroblasts and epidermal cells tolerate hypoxia for longer periods, neurons undergo irreversible damage within minutes of oxygen deprivation.

128. (D) Fibrinogen
During the process of primary hemostasis, platelet aggregation is mediated by the binding of specific receptors. Activated platelets express glycoprotein IIb/IIIa receptors on their membranes, which bind to circulating fibrinogen molecules. Fibrinogen acts as a bridge, linking multiple platelets together to form the primary platelet plug, which temporarily seals the vascular defect.

129. (A) Decreased capillary hydrostatic pressure
Virchow's triad outlines the three primary pathophysiological categories that contribute to thrombus formation. These factors include endothelial cell injury, alterations in normal blood flow such as stasis or turbulence, and systemic hypercoagulability. Decreased capillary hydrostatic pressure does not promote thrombosis; instead, it reduces fluid extravasation, remaining completely unrelated to Virchow's triad.

130. (A) P-3, Q-4, R-1, S-2
Pathological metabolic disturbances manifest in characteristic histological ways. Disrupted protein metabolism can lead to hyaline change, while abnormal lipid metabolism results in steatosis or fatty change. Defective carbohydrate metabolism underlies the glycogenoses, and altered purine or uric acid metabolism causes gout. These classic associations define key histopathological patterns observed in affected tissues.

NODIA

131. (A) Thrombi formed in flowing blood
The lines of Zahn are distinctive microscopic and macroscopic bands characterized by alternating pale layers of platelets and fibrin with darker layers of erythrocytes. These lines only form when a thrombus develops within actively flowing blood, making them highly diagnostic for antemortem thrombi and helping distinguish them from postmortem blood clots.

132. (D) Cardiopulmonary resuscitation resulting in rib fractures
Bone marrow embolism occurs when marrow tissue, containing hematopoietic cells and fat, enters injured venous sinusoids. This condition is most frequently observed as a complication of severe skeletal trauma, particularly rib and sternal fractures resulting from vigorous cardiopulmonary resuscitation, rather than soft tissue burns, venous catheter displacement, or disseminated intravascular coagulation.

133. (B) Coagulative necrosis
Chronic, sublethal ischemia allows cells to adapt to reduced oxygen and nutrient levels. This state typically leads to adaptive changes, including cellular atrophy, decreased cell size, and fatty change. Coagulative necrosis, however, represents irreversible cell death, which is not an adaptive response to sublethal stress but rather the outcome of acute, lethal ischemia.

134. (C) Erythrocyte rouleaux formation
Sludged blood involves abnormal aggregation of red blood cells, primarily driven by the formation of erythrocyte rouleaux. Elevated levels of large plasma proteins, such as fibrinogen and globulins, reduce the negative electrical charge on erythrocytes. This charge reduction allows them to stack together, increasing blood viscosity and impairing microvascular perfusion in tissue beds.

135. (A) I, II, III, IV
The intrinsic apoptotic pathway begins with mitochondrial outer membrane permeabilization, releasing cytochrome c into the cytosol. Cytochrome c binds to Apaf-one to assemble the wheel-like apoptosome. This complex recruits and activates initiator Caspase-nine, which then enzymatically cleaves and activates executioner caspases, such as Caspase-three, to complete the program of cell death.

136. (A) Cellular injury causing multi-organ failure despite resuscitation
The irreversible stage of shock is defined by severe, widespread cellular and tissue injury that has progressed to a point where therapeutic intervention can no longer save life. Even if hemodynamic parameters are temporarily restored, catastrophic cell damage, lysosomal enzyme leakage, and multi-organ failure inevitably result in death, making resuscitation efforts unsuccessful.

137. (B) Hydropic degeneration
Hydropic degeneration, or vacuolar degeneration, is characterized by the accumulation of water within the cytoplasm of cells, resulting in a swollen, vacuolated appearance. In the renal tubule epithelium, exposure to hypertonic osmotic agents or severe hypokalemia disrupts active ion transport, causing massive water influx and severe, reversible vacuolar changes in the cells.

138. (B) Gout
Gout is a metabolic disorder characterized by hyperuricemia, which stems from disturbances in purine metabolism. When uric acid levels exceed solubility limits, monosodium urate crystals precipitate within joints and soft tissues, triggering acute and chronic inflammatory arthritis. This pathology directly represents a classic metabolic error in the breakdown of purine nucleotides in the body.

139. (A) Spleen
Spleen involvement in amyloidosis classically presents in two distinct macroscopic patterns. The sago spleen pattern features amyloid deposits limited to the splenic follicles, resembling glistening tapioca grains. In contrast, the lardaceous spleen pattern involves diffuse amyloid deposition throughout the red pulp, giving the entire organ a firm, waxy, map-like appearance under gross examination.

140. (C) A is true but R is false.
Lipofuscin is an insoluble, yellow-brown pigment that accumulates in aging cells, particularly in the heart, liver, and brain. It is considered an innocuous byproduct of lipid peroxidation of subcellular membranes and does not cause cell necrosis or disrupt lysosomes, making the assertion true but the accompanying reason completely false in its pathophysiological description.

141. (D) Excessive mobilization of peripheral free fatty acids
Starvation triggers a massive lipolysis in peripheral adipose tissues due to low insulin and elevated glucagon levels. This response releases abundant free fatty acids into the circulation, which are taken up by the liver. The liver is overwhelmed by this influx, leading to excessive triglyceride synthesis and subsequent hepatic steatosis or fatty change.

142. (C) Carbohydrate metabolism
Diabetic microangiopathy, characterized by diffuse thickening of capillary basement membranes, is a chronic complication of diabetes mellitus, a primary disorder of carbohydrate metabolism. Persistent hyperglycemia leads to non-enzymatic glycation of proteins and abnormal basement membrane matrix production, impairing vascular function and causing widespread organ damage throughout the vascular tree.

Click Here to Buy book on Flipkart

143. (B) Albinism
Albinism is an inherited disorder characterized by a complete or partial deficiency of melanin synthesis in the melanocytes. This condition is most commonly caused by a genetic mutation resulting in a deficiency of the enzyme tyrosinase, which is essential for converting tyrosine into melanin precursors, leaving the skin, hair, and eyes unpigmented.

144. (D) An enzymatic defect in the heme biosynthetic pathway
Porphyrias are a group of inherited or acquired metabolic disorders characterized by defects in specific enzymes of the heme biosynthetic pathway. These enzymatic blocks lead to the abnormal accumulation and excretion of porphyrins or their precursors, which are highly reactive and cause cutaneous photosensitivity, neurological dysfunction, and visceral pain in affected individuals.

145. (A) Cotton dust
Byssinosis is an occupational lung disease, classified as a pneumoconiosis, caused by the chronic inhalation of cotton, flax, or hemp dust. It typically occurs in textile mill workers, presenting with chest tightness and bronchoconstriction, characteristically worse on the first day of the workweek, earning the common name of Monday chest tightness.

146. (C) Mitochondrial calcium entry and binding to denatured proteins
Dystrophic calcification initiates in necrotic tissues when dying cells lose their ability to regulate intracellular calcium. Calcium ions accumulate within the mitochondria, binding to membrane phospholipids and denatured proteins. This localized calcium concentration serves as a nucleation site for phosphate binding, leading to the crystallization of calcium phosphate in damaged tissues.

147. (A) Bax
Bax is a key pro-apoptotic member of the Bcl-two family of proteins. Under apoptotic stimuli, Bax undergoes conformational changes, oligomerizes, and inserts into the outer mitochondrial membrane. This action creates pores that increase membrane permeabilization, allowing the release of cytochrome c and other pro-apoptotic proteins into the cytosol to trigger cell death.

148. (D) Preservation of the cellular outline but loss of nuclear details
Coagulative necrosis is morphologically characterized by the preservation of the basic structural outline of the dead cells and tissues for several days. Although the cells exhibit eosinophilic cytoplasm and lose their nuclear details through karyolysis, pyknosis, or karyorrhexis, their overall cellular ghost outline remains recognizable under light microscopy in affected tissues.

149. (B) Volvulus or intussusception causing vascular occlusion
Moist gangrene occurs in organs with a rich blood supply and abundant moisture, such as the intestines. It typically results from sudden venous occlusion, as seen in volvulus or intussusception. The blocked outflow leads to intense congestion, tissue necrosis, and rapid invasion by putrefactive bacteria, causing swollen, foul-smelling, black tissue in the gut.

150. (A) I, II, III and IV
Hemostasis and thrombosis are regulated by balanced pathways. Thromboxane A-two and von Willebrand factor promote platelet aggregation and adhesion, while prostacyclin inhibits it. Plasmin degrades fibrin clots into D-dimers. Antithrombin three, however, directly inhibits thrombin rather than activating it, making the first four listed statements correct in this clinical context.

151. (B) Karyolysis
Karyolysis refers to the progressive enzymatic dissolution of the cell nucleus during necrotic cell death. As lysosomal DNases degrade the nuclear DNA, the basophilic chromatin fades and eventually disappears entirely, leaving only a pale nuclear ghost or a completely empty cytoplasm. This process is distinct from pyknosis and karyorrhexis in necrotic cells.

152. (A) Continuous fluid drainage by the lymphatic system
Under normal physiological conditions, a small amount of fluid continuously filters out of capillaries into the interstitial space. This fluid is actively and continuously returned to the systemic circulation by the lymphatic system. Effective lymphatic drainage prevents fluid accumulation, maintaining a dry interstitium

Downloaded from www.pdf.tube

and preventing the development of edema in healthy tissues.

153. (B) Severe ascites and chronic hepatic congestion
Right-sided heart failure impairs the heart's ability to pump blood from the systemic venous system into the lungs. This failure leads to systemic venous congestion, which is most prominent in the portal and hepatic circulations. The resulting elevated hydrostatic pressure causes chronic passive hepatic congestion and transudative fluid accumulation, producing severe ascites.

154. (B) Lactic acid
Hypoxia impairs oxidative phosphorylation, forcing cells to switch to anaerobic glycolysis to generate ATP. This metabolic shift increases the rate of glycogen breakdown and leads to the overproduction of lactic acid and inorganic phosphates. The accumulation of lactic acid decreases intracellular pH, causing chromatin clumping and enzyme inactivation within the cell.

155. (A) Primary haemostatic defect such as thrombocytopenia
Petechiae are tiny, pinpoint hemorrhages measuring one to two millimeters on skin or mucosal surfaces. They are classically indicative of primary hemostatic defects, such as a severe reduction in platelet count or defective platelet function. In contrast, coagulation factor deficiencies typically present with larger ecchymoses or hematomas in the deep tissues.

156. (C) Exposure of factor XII to a negatively charged surface
The intrinsic pathway of blood coagulation, or contact activation pathway, is initiated in vitro when circulating Factor twelve comes into contact with a negatively charged surface, such as glass or subendothelial collagen. This interaction triggers a cascade of sequential zymogen activations, eventually leading to thrombin generation and the formation of a fibrin clot.

157. (D) Pulmonary arteries
Thrombi that originate in the deep veins of the lower extremities travel through the vena cava, enter the right atrium and ventricle, and are pumped into the pulmonary arterial tree. Consequently, these emboli most commonly lodge in the pulmonary arteries, potentially causing a life-threatening pulmonary embolism and severe cardiorespiratory compromise.

158. (C) Lack of collateral blood flow preventing necrotic area haemorrhage
White or anemic infarcts occur in solid organs with end-arterial circulations, such as the kidney, spleen, and heart. When the single feeding artery is occluded, the lack of collateral blood flow prevents blood from entering the necrotic zone. The tissue remains pale, and subsequent swelling squeezes remaining blood out of the region.

159. (B) The ratio of plasma globulins and fibrinogen to albumin
The reversibility of sludged blood is heavily dependent on the ratio of asymmetric plasma proteins, like globulins and fibrinogen, to albumin. High levels of globulins and fibrinogen decrease the negative surface charge of erythrocytes, promoting aggregation. Sufficient albumin levels help maintain charge repulsion, keeping cells dispersed and preventing stasis in the microcirculation.

NODIA

160. (D) It features high cardiac output and massive systemic vasodilation
Cardiogenic shock is characterized by a profound reduction in cardiac output due to primary myocardial pump failure, such as from myocardial infarction or tamponade. To compensate for this low output, the sympathetic nervous system triggers systemic vasoconstriction, making high cardiac output and massive vasodilation clinically incorrect features for this type of shock.

161. (A) Liver and kidneys
Hydropic degeneration is an early, reversible response to cellular injury, occurring when ATP depletion impairs the sodium-potassium ATPase pump. Highly metabolic cells, such as hepatocytes in the liver and tubular epithelial cells in the kidneys, are rich in ATP-driven pumps and are therefore most sensitive to this swelling in injury states.

162. (C) Descriptive, morphologic term for glassy, eosinophilic material
Hyaline change is a broad, descriptive histological term rather than a specific biochemical entity. It refers to any intracellular or extracellular alteration that gives a glassy, homogeneous, bright pink appearance in hematoxylin and eosin stained tissue sections. It can represent

accumulated immunoglobulins, plasma proteins, or degenerated collagen in various pathological processes.

163. (A) Apple-green birefringence under polarized light with Congo red
Amyloid proteins contain a unique beta-pleated sheet conformation that binds specifically to Congo red dye molecules. When viewed under polarized light microscopy, this structured binding produces a pathognomonic apple-green birefringence. This unique optical property is the gold standard for distinguishing amyloid from other hyaline depositions in various tissue sections.

164. (D) Prolonged anorexia triggering massive lipid mobilization
Feline hepatic lipidosis, or fatty liver, is a severe metabolic syndrome classically triggered by prolonged anorexia in obese cats. The lack of food intake induces a state of starvation, driving massive lipolysis in peripheral adipose tissues. This mobilization floods the hepatocytes with free fatty acids, which accumulate as triglycerides due to impaired metabolic processing.

165. (B) Diastase digestion to enzymatically remove the glycogen
Glycogen deposits stain bright magenta with the Periodic Acid-Schiff reagent. To confirm that this staining is due to glycogen rather than other glycoproteins, a parallel tissue section is treated with diastase, an enzyme that specifically digests glycogen. A loss of magenta staining in the treated section confirms the presence of glycogen in tissues.

166. (C) Is associated with pathological states rather than simple aging
Ceroid is an autofluorescent, yellow-brown lipid pigment that is histochemically similar to lipofuscin but differs in its clinical context. While lipofuscin is a physiological byproduct of wear-and-tear in aging cells, ceroid accumulates rapidly in pathological states, such as severe vitamin E deficiency, malnutrition, or specific inherited lysosomal storage disorders in animals.

167. (C) 2.0 to 2.5 mg/dL
Jaundice, or icterus, is the yellowish discoloration of the skin, sclera, and mucous membranes caused by tissue deposition of bile pigments. Normal serum bilirubin levels are below one milligram per deciliter. Jaundice only becomes clinically detectable when total serum bilirubin concentration exceeds two point zero to two point five milligrams per deciliter.

168. (A) Silicosis
Pneumoconioses are a group of chronic interstitial lung diseases caused by the inhalation of inorganic dusts, mineral fibers, or chemical particles. Silicosis, caused by the inhalation of crystalline silica dust, is a classic example of this condition, inducing a fibrogenic response that leads to progressive pulmonary fibrosis and nodule formation in lungs.

169. (B) Hypercalcaemia secondary to hyperparathyroidism or bone damage
Metastatic calcification occurs when calcium deposits in normal, healthy tissues due to systemic hypercalcemia or altered calcium-phosphate metabolism. This systemic imbalance is typically secondary to conditions like hyperparathyroidism, vitamin D toxicity, renal failure, or extensive bone destruction from osteolytic tumors, rather than localized tissue damage, cellular necrosis, or ischemic cardiac infarction.

170. (A) P-3, Q-2, R-1, S-4
Different necrosis types present distinct histopathological features. Fibrinoid necrosis shows intense eosinophilic deposits in vessel walls, while fat necrosis undergoes saponification, leaving chalky white deposits. Liquefactive necrosis involves enzymatically digested tissue forming a viscous liquid, and coagulative necrosis preserves cell outlines despite the loss of nuclei in affected tissues.

171. (A) Coagulative necrosis with desiccation
Dry gangrene is a clinical variant of coagulative necrosis that develops in distal extremities when arterial blood supply is critically compromised. The affected tissue undergoes ischemic necrosis and subsequent desiccation, turning dry, shriveled, and dark due to hemoglobin breakdown, without significant bacterial putrefaction or liquefaction occurring in the necrotic tissues.

172. (C) A reversible shift to decrease protein synthesis and conserve energy
During early cell injury, the dissociation of ribosomes from the rough endoplasmic reticulum and the swelling of cisternae are classic,

Click Here to Buy book on Amazon

reversible changes. This dissociation serves as an adaptive, energy-conserving response, allowing the cell to downregulate energy-intensive protein synthesis and focus remaining ATP resources on maintaining vital survival pathways during cellular stress.

173. (D) Released lysosomal enzymes digest essential cellular macromolecules
Lysosomes contain a variety of acid hydrolases, including nucleases, proteases, and lipases, which are highly destructive. The rupture of lysosomal membranes releases these active enzymes into the cytosol. Once in the cytoplasm, these enzymes uncontrollably digest essential proteins, nucleic acids, and lipids, driving the cell into irreversible injury and autolytic necrotic cell death.

174. (C) Massive calcium influx causing permanent mitochondrial damage
The point of no return in ischemic myocardial injury is defined by the massive influx of extracellular calcium across damaged membranes. This uncontrolled calcium surge activates destructive intracellular enzymes and deposits as amorphous densities in mitochondria, permanently disrupting oxidative phosphorylation and preventing any recovery of cell viability in the damaged heart.

175. (B) Ascites
Ascites is the clinical term used to describe the abnormal accumulation of transudative fluid within the peritoneal cavity. This condition is commonly driven by increased hydrostatic pressure in the portal venous system, as seen in liver cirrhosis, or decreased plasma oncotic pressure, resulting in excessive fluid extravasation into the abdominal cavity.

176. (D) Left-sided heart failure leading to pulmonary capillary engorgement
Chronic passive congestion of the lungs is driven by left-sided heart failure. When the left ventricle fails to pump blood effectively, pressure backs up into the left atrium and pulmonary veins. This backup causes chronic engorgement of pulmonary capillaries, leading to micro-hemorrhages and the accumulation of hemosiderin-laden macrophages in the alveoli.

177. (A) Impaired removal of metabolic waste products such as lactic acid
Ischemia differs fundamentally from hypoxia because it involves a reduction or complete cessation of blood flow, rather than just oxygen deprivation. Consequently, ischemia not only deprives tissues of oxygen and glycolytic substrates but also impairs the removal of toxic metabolic waste products, such as lactic acid, accelerating progressive cellular damage and injury.

178. (C) A localized collection of blood outside the blood vessels
A hematoma is defined as a localized accumulation of blood within a tissue or anatomical space outside the blood vessels. This accumulation usually results from vascular injury, allowing blood to pool and clot. Hematomas can range from small, relatively harmless subcutaneous bruises to life-threatening internal collections in the cranium or major organs.

NODIA

179. (A) Is attached to the cardiac wall or large arteries without fully occluding the lumen
A mural thrombus is a non-occlusive thrombus that adheres to the wall of a large blood vessel or a cardiac chamber, such as the left ventricle or aorta. Although they do not completely block blood flow through these wide channels, they present a significant clinical risk by potentially embolizing to distal arterial vascular beds.

180. (A) Both A and R are true and R is the correct explanation of A.
Irreversible cell injury is fundamentally defined by permanent mitochondrial dysfunction and profound cell membrane damage. Membrane rupture allows a massive, uncontrolled influx of extracellular calcium, which activates destructive intracellular enzymes that degrade structural proteins and genomic DNA, explaining why membrane failure inevitably seals the cell's demise in necrotic pathways.

181. (B) An arteriovenous shunt or patent interatrial septal defect
A paradoxical embolism occurs when a thrombus originating in the venous system bypasses the pulmonary circulation to enter the systemic arterial tree. This passage requires an abnormal anatomical communication between the right and left sides of the heart or circulation, such as

a patent interatrial septal defect or an abnormal arteriovenous shunt.

182. (C) Lungs and intestines
Red or hemorrhagic infarcts occur in tissues with a dual blood supply, such as the lungs, or loose tissues that allow blood to pool, such as the intestines. When arterial occlusion occurs, collateral vessels or dual circulations continue to pump blood into the necrotic zone, causing extensive hemorrhage into the damaged tissue parenchyma.

183. (D) It leads to micro-infarctions and severe tissue ischaemia
Sludged blood increases microvascular flow resistance due to cellular aggregation, reducing local perfusion. This stasis and viscosity increase directly lead to the occlusion of capillaries and small venules. The resulting lack of nutrient and oxygen delivery causes localized micro-infarctions, tissue ischemia, and potential organ dysfunction throughout the affected tissue beds.

184. (A) Lipopolysaccharide endotoxin
In septic shock caused by gram-negative bacteria, lipopolysaccharide from the bacterial outer membrane is the primary trigger. Lipopolysaccharide binds to circulating proteins and toll-like receptors on immune cells, initiating a massive release of pro-inflammatory cytokines, which leads to widespread endothelial damage, systemic vasodilation, and severe circulatory collapse in the body.

185. (B) Distended segments of the endoplasmic reticulum
During cellular swelling, or hydropic degeneration, ATP depletion impairs active sodium-potassium pumps. The resulting influx of sodium and water causes water to accumulate within the cell. Histologically, this water collects within the cisternae of the endoplasmic reticulum, causing them to dilate and appear as clear, fluid-filled cytoplasmic vacuoles under light microscopy.

186. (B) Hepatocellular alpha-1 antitrypsin deficiency
Alpha-one antitrypsin deficiency is a genetic disorder where a mutation leads to the misfolding of the alpha-one antitrypsin protein within the liver. The misfolded proteins cannot be secreted and instead aggregate inside the endoplasmic reticulum of hepatocytes, forming round, eosinophilic, PAS-positive globules that cause significant cellular stress and liver injury.

187. (A) Associated with plasma cell dyscrasias producing AL amyloid
Primary amyloidosis is a systemic disease associated with plasma cell dyscrasias, such as multiple myeloma. In this condition, clonal populations of plasma cells produce excessive monoclonal immunoglobulin light chains. These light chains are processed and deposit in tissues as amyloid light fibrils, leading to progressive organ dysfunction and failure in affected patients.

188. (C) Accumulation of adipocytes in interstitial connective tissue
Fatty infiltration is characterized by the abnormal presence and accumulation of mature adipocytes within the interstitial connective tissue of an organ, such as the heart or pancreas. This condition is distinct from fatty change or steatosis, which refers to the abnormal accumulation of triglycerides within the cytoplasm of parenchymal cells themselves.

189. (D) Diabetic nephropathy inducing tubular glycogen accumulation
Armanni-Ebstein lesions are a classic histopathological finding in poorly controlled diabetes mellitus. They are characterized by severe glycogen accumulation within the epithelial cells of the renal tubules, particularly in the straight portion of the proximal tubule. This glycogen deposition occurs in response to severe glycosuria and glucose reabsorption in the kidneys.

190. (B) I, II and V
Dry gangrene typically affects the distal extremities, not internal organs, which instead undergo moist gangrene. Moist gangrene is always modified by active putrefactive bacterial infection. Gangrene inherently begins as coagulative necrosis before being modified, making statements one, two, and five incorrect in describing the characteristics of these gangrenous processes.

191. (B) The porphyrin ring of heme
Bilirubin is an endogenous pigment produced during the degradation of senescent red blood cells. Inside macrophages of the reticuloendothelial system, hemoglobin is broken down. The globin chains are recycled, and iron is salvaged, while

Downloaded from www.pdf.tube

the remaining protoporphyrin ring of the heme moiety is enzymatically converted into biliverdin and then into free bilirubin.

192. (D) Alveolar macrophages secreting pro-fibrogenic cytokine molecules
In silicosis, inhaled crystalline silica particles are phagocytosed by alveolar macrophages. Because silica is highly cytotoxic, it disrupts lysosomal membranes, killing the macrophages and releasing silica back into the tissue. This cycle drives macrophages to continuously secrete pro-inflammatory and pro-fibrogenic cytokines, such as TGF-beta, stimulating intense fibroblast proliferation and lung fibrosis.

193. (A) Normal systemic calcium levels and localized cellular damage or necrosis
Dystrophic calcification is the pathological deposition of calcium salts in injured, dying, or necrotic tissues. This process occurs locally despite completely normal systemic calcium levels and in the absence of any generalized calcium metabolic abnormalities, distinguishing it from metastatic calcification, which requires systemic hypercalcemia to deposit calcium in healthy tissues.

194. (C) Involves cell shrinkage rather than cell swelling
Apoptosis is a highly regulated, active process of programmed cell death characterized by cell shrinkage, chromatin condensation, and the formation of apoptotic bodies without membrane rupture. This process contrasts directly with necrosis, which is a passive, pathological death marked by cellular swelling, membrane rupture, and a subsequent acute inflammatory tissue response.

195. (A) Presence of lipid-laden gitter cells and cystic tissue spaces
Liquefactive necrosis in the brain is characterized by the rapid enzymatic dissolution of necrotic neural tissue. As the tissue liquefies, specialized macrophages called gitter cells migrate to the area to phagocytose lipid-rich myelin breakdown products. This process eventually leaves behind a fluid-filled cystic cavity surrounded by reactive glial cells in the brain.

NODIA

196. (D) Alpha toxin lecithinase released by Clostridium perfringens
Gas gangrene, or clostridial myonecrosis, is a life-threatening infection caused primarily by Clostridium perfringens. The bacteria release several potent exotoxins, most notably alpha toxin, a lecithinase that degrades cell membrane phospholipids. This enzymatic action causes widespread myonecrosis, hemolysis, and gas production, leading to tissue crepitus and severe systemic shock.

197. (B) Loss of cristae and severe mitochondrial membrane disruption
The transition from reversible to irreversible cell injury is marked by profound mitochondrial damage. Ultrasturally, this damage includes the loss of normal mitochondrial cristae, high-amplitude swelling, and the disruption of the outer and inner mitochondrial membranes. This disruption permanently halts oxidative phosphorylation, preventing any possibility of cell survival in injured tissue.

198. (C) Calcium-dependent proteases like calpains
During cell injury, a rise in cytosolic calcium levels activates a variety of destructive enzymes. Among these, calcium-dependent neutral proteases, known as calpains, are activated. Calpains degrade key structural cytoskeletal proteins, such as spectrin and tubulin, disrupting the structural integrity of the cell membrane and contributing to blebbing and cell death.

199. (D) Severe, generalized massive oedema with profound tissue swelling
Anasarca is a clinical term that describes severe, generalized, and widespread edema. It is characterized by profound subcutaneous swelling throughout the entire body, often accompanied by fluid accumulation within major serous cavities, such as the pleural, pericardial, and peritoneal spaces, typically resulting from severe cardiac, renal, or hepatic organ failure.

200. (A) I, III, IV and V
Steatosis represents an absolute increase in intracellular lipids, which is a reversible process rather than invariably leading to cell death. Alcohol abuse, hypoxia, and genetic disorders like Niemann-Pick disease are well-established causes or examples of lipid metabolism disturbances, making statements one, three, four, and five correct in this metabolic context.

201. (B) Liver
Nutmeg liver is a classic gross pathological description of chronic passive congestion of the liver, typically caused by right-sided heart failure. The congestion leads to blood pooling in the centrilobular zones, which appear dark red, surrounded by pale, fatty periportal hepatocytes. This alternating pattern resembles the cut surface of a nutmeg.

202. (B) Chronic passive congestion of pulmonary tissues
In chronic passive congestion of the lungs, elevated pulmonary venous pressure causes blood to pool in the alveolar capillaries. Some capillaries rupture, releasing red blood cells into the alveoli. Alveolar macrophages phagocytose these cells and convert the hemoglobin into hemosiderin, producing characteristic hemosiderin-laden macrophages that indicate chronic congestion in tissues.

203. (A) Histotoxic hypoxia
Histotoxic hypoxia occurs when cells are fully supplied with oxygen but are unable to utilize it due to the inhibition of cellular respiratory enzymes. Cyanide poisoning is the classic example, where cyanide binds to and inhibits cytochrome c oxidase in the mitochondrial electron transport chain, blocking ATP production despite adequate oxygen delivery.

204. (C) A primary platelet plug
Primary hemostasis is the initial response to vascular injury, involving platelet adhesion, activation, and aggregation. This process culminates in the formation of a temporary, primary platelet plug at the site of endothelial damage. This fragile plug is later reinforced and stabilized by a mature fibrin mesh during secondary hemostasis.

205. (D) Prostacyclin and nitric oxide
Intact, healthy endothelial cells actively maintain blood fluidity by inhibiting platelet activation and coagulation. The primary mechanism of this anticoagulant effect is the continuous synthesis and secretion of nitric oxide and prostacyclin. These potent molecules dilate blood vessels and act as strong physiological inhibitors of platelet adhesion and aggregation in circulation.

206. (C) Fat embolism
Fat embolism syndrome is characteristically triggered by severe skeletal trauma, particularly fractures of long bones like the femur, or extensive damage to adipose tissues. This physical disruption allows microscopic fat droplets from the yellow bone marrow or ruptured adipocytes to enter damaged venous sinusoids, embolizing to the lungs and systemic circulation.

207. (B) Depressed, pale fibrotic scar
In solid organs like the kidney or spleen, a healed infarct is characterized by a depressed, pale fibrotic scar. Because these organs undergo coagulative necrosis, the dead tissue is slowly removed by phagocytes and replaced by collagenous connective tissue, which contracts over time, leaving a permanent structural depression on the organ surface.

208. (D) Blood viscosity in the microcirculation
Sludged blood impairs tissue perfusion primarily by increasing blood viscosity within the microcirculation. The aggregation of red blood cells into clusters blocks the smooth flow of blood through capillaries and venules, dramatically raising resistance to flow, causing capillary stasis, and leading to localized tissue hypoxia and severe microvascular infarctions.

209. (A) Widespread tissue hypoxia and metabolic lactic acidosis
In the progressive stage of shock, compensatory mechanisms fail to maintain adequate perfusion. This failure leads to widespread tissue hypoperfusion, forcing cells throughout the body to shift to anaerobic metabolism. This shift results in profound tissue hypoxia, cellular injury, and the accumulation of lactic acid, causing severe metabolic lactic acidosis.

210. (C) Intracellular potassium loss and gain of sodium and water
Reversibility of cell injury is fundamentally characterized by the failure of energy-dependent sodium-potassium pumps. This failure leads to a loss of intracellular potassium and an accumulation of intracellular sodium. The osmotic gradient created by the excess sodium drives water into the cell, resulting in early cellular swelling, also known as hydropic change.

****

Click Here to Buy book on Flipkart

CHAPTER 03
Inflammation and Healing

NOOBIA

2012

1. Which of the following best defines the primary physiological purpose of acute inflammation in response to tissue injury?
(A) To immediately initiate the proliferation of fibroblasts and extracellular matrix deposition
(B) To dilute, isolate, and eliminate the inciting agent and prepare the tissue for repair
(C) To trigger an adaptive immune response primarily through naive T-cell activation
(D) To induce apoptosis in surrounding healthy cells to prevent the spread of infection

2022

2. Which type of necrotic stimulus is most frequently recognized by Toll-like receptors and NOD-like receptors to initiate sterile inflammation?
(A) Damage-associated molecular patterns
(B) Pathogen-associated molecular patterns
(C) Complement decay-accelerating factors
(D) Major histocompatibility complex class II

2013

3. Identify the correct statement regarding physical triggers of acute inflammation.
(A) Ionizing radiation induces inflammation primarily through localized cellular disruption and direct complement activation.
(B) Thermal burns suppress the local arachidonic acid cascade, leading to delayed and weakened cellular responses.
(C) Mechanical trauma initiates inflammation through rapid histamine release from resident tissue mast cells.
(D) Physical injury triggers acute tissue inflammation exclusively via the activation of the kinin-kallikrein pathway.

2022

4. Identify the INCORRECT statement regarding the immunological triggers of inflammation.
(A) Type I hypersensitivity reactions involve IgE-mediated mast cell degranulation and subsequent release of vasoactive amines.
(B) Immune complex deposition in tissues activates the classical complement pathway, leading to chemotactic factor generation.
(C) Cell-mediated hypersensitivity relies on cytokine release from sensitized T lymphocytes to recruit and activate macrophages.
(D) Autoimmune reactions bypass acute phases to directly initiate granulomatous inflammation.

2025

5. What is the direct effect of strong alkaline chemical exposure on tissue that leads to an intense inflammatory response?
(A) Coagulative necrosis with preservation of basic tissue architecture for several days
(B) Caseous necrosis with formation of a structured and well-defined granuloma
(C) Liquefactive necrosis with cellular dissolution and lipid breakdown
(D) Fibrinoid necrosis limited strictly to the endothelial lining of small vessels

2017

6. A researcher is studying an infectious trigger of inflammation. Which of the following exogenous molecules is most likely to act as a potent trigger for the inflammatory cascade via Toll-like receptor 4?
(A) Peptidoglycan
(B) Lipopolysaccharide
(C) Zymosan
(D) Flagellin

2012

7. According to etiology, how is inflammation triggered by retained surgical sutures typically classified?
(A) Infectious inflammation
(B) Immunological hypersensitivity
(C) Necrotic sterile inflammation
(D) Foreign body inflammation

2004

8. In comparing infectious and physical triggers of inflammation, which feature is unique to infectious triggers?
(A) The elaboration of pathogen-associated

molecular patterns
(B) The generation of reactive oxygen species
(C) The synthesis of prostaglandins
(D) The release of intracellular damage-associated molecular patterns

2002
9. Which of the following cardinal signs of inflammation is NOT primarily caused by local hemodynamic changes and increased vascular permeability?
(A) Calor (heat)
(B) Rubor (redness)
(C) Functio laesa
(D) Tumor (swelling)

2024
10. Assess the following assertion and reason regarding the cardinal signs of inflammation, and choose the correct option.
Assertion (A): The cardinal sign "calor" (heat) is typically observed only in peripheral skin inflammations, not in deep internal organ inflammations.
Reason (R): Deep internal organs are already at core body temperature, so local vasodilation does not significantly raise their temperature relative to surrounding tissues.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2025
11. Which of the following chemical mediators is most directly responsible for the cardinal sign "dolor" (pain) during acute inflammation?
(A) Leukotriene B_4
(B) Interleukin-8
(C) Thromboxane A_2
(D) Bradykinin

2005
12. From the observation of "tumor" (swelling) at an inflammatory site, what can be accurately inferred regarding the local microvasculature?
(A) There is a profound decrease in localized capillary hydrostatic pressure.
(B) Protein-rich exudate accumulates in the extravascular space.
(C) Endothelial cell junctions have tightened to retain intravascular fluid.
(D) Lymphatic drainage from the tissue has completely ceased functioning.

2010
13. Which of the following best describes the physiological advantage of the cardinal sign "functio laesa" (loss of function) in the context of an acutely inflamed joint?
(A) It actively accelerates the dissemination of pyogenic bacteria throughout the local joint capsule.
(B) It promotes immediate, extensive collagen deposition and scar tissue formation by disabling movement.
(C) It encourages host rest of the injured part to prevent further damage.
(D) It upregulates the rapid enzymatic degradation of articular cartilage to clear the joint space.

2011
14. What is the anatomical basis for the cardinal sign "rubor" (redness) observed in acute inflammation?
(A) Accumulation of desquamated epithelial cells
(B) Extravasation of red blood cells into the interstitial tissue
(C) Deposition of fibrin networks within the venules
(D) Arteriolar vasodilation and engorgement of capillary beds

2009
15. Who was the Roman scholar responsible for first describing the four classic signs of inflammation (rubor, tumor, calor, dolor)?
(A) Aulus Cornelius Celsus
(B) Hippocrates of Kos
(C) Claudius Galenus
(D) Rudolf Ludwig Carl Virchow

2025
16. Identify the correct statement regarding the fifth cardinal sign of inflammation.
(A) Functio laesa was originally proposed by Aulus Cornelius Celsus during the first century.
(B) Functio laesa was added to the signs by Rudolf Virchow in the 19th century.
(C) Functio laesa refers specifically to the loss of biochemical enzymatic function in lysosomes.
(D) Functio laesa occurs strictly and exclusively as a result of ischemic tissue necrosis.

NODIA

2011

17. Identify the INCORRECT statement regarding the hemodynamic changes observed in acute inflammation.
(A) Transient vasoconstriction of arterioles often precedes vasodilation.
(B) Increased vascular permeability invariably occurs before any arteriolar vasodilation.
(C) Vasodilation leads to increased blood flow, causing heat and redness.
(D) Stasis of blood flow results from the loss of intravascular fluid and concentration of red blood cells.

2025

18. What is the primary physical cause of blood flow stasis in the microcirculation during acute inflammation?
(A) Increased blood viscosity from exudation of protein-rich fluid
(B) Systemic hypercoagulability leading to widespread, occlusive capillary microthrombi
(C) Direct, mechanical compression of post-capillary venules by proliferating fibroblasts
(D) Rapid, widespread apoptosis of endothelial cells lining the microvascular arterioles

2022

19. A patient presents with acute appendicitis. Histological examination reveals engorged capillaries packed with erythrocytes and margination of leukocytes. What principle of fluid dynamics explains the slowing of blood flow that allows this margination?
(A) Bernoulli's principle of fluid dynamics in constricted tubes
(B) Starling's law governing cardiac output and stroke volume
(C) Poiseuille's law on viscosity and flow rate
(D) Laplace's law of wall tension in hollow spherical organs

2001

20. Match the hemodynamic changes in List I with their corresponding primary mechanisms in List II:

List I: Hemodynamic Change
P. Transient vasoconstriction
Q. Vasodilation
R. Increased permeability
S. Stasis

List II: Primary Mechanism
1. Endothelial cell contraction
2. Hemoconcentration and increased viscosity
3. Neurogenic reflex
4. Histamine acting on vascular smooth muscle

(A) P-4, Q-3, R-2, S-1
(B) P-1, Q-2, R-3, S-4
(C) P-3, Q-1, R-4, S-2
(D) P-3, Q-4, R-1, S-2

2022

21. How is the initial vascular response to a mild thermal injury primarily classified in terms of duration and onset?
(A) Delayed prolonged response
(B) Immediate sustained response
(C) Immediate transient response
(D) Biphasic sustained response

2019

22. When comparing the hydrostatic and osmotic pressures in an acutely inflamed capillary bed to a normal capillary bed, which of the following is true?
(A) Inflamed beds have lower intravascular hydrostatic pressure and lower interstitial osmotic pressure.
(B) Inflamed beds have higher intravascular hydrostatic pressure and higher interstitial osmotic pressure.
(C) Inflamed beds have lower intravascular hydrostatic pressure and higher interstitial osmotic pressure.
(D) Inflamed beds have higher intravascular hydrostatic pressure and lower interstitial osmotic pressure.

2016

23. Which of the following is an exception to the rule that vasodilation affects the entire microvascular bed in acute inflammation?
(A) Capillaries, which completely lack smooth muscle and are forced to dilate passively
(B) Post-capillary venules, which undergo active, receptor-mediated smooth muscle relaxation
(C) Arterioles, which primarily dictate the downstream microvascular inflow into the bed
(D) Large muscular arteries, which do not dilate during localized inflammation

2002

24. Which sequence of microvascular events best describes the immediate hemodynamic response to an acute tissue injury?
(A) Transient vasoconstriction, vasodilation, increased permeability, stasis
(B) Stasis, transient vasoconstriction, vasodilation, increased permeability

Downloaded from www.pdf.tube

(C) Vasodilation, stasis, transient vasoconstriction, increased permeability
(D) Increased permeability, transient vasoconstriction, stasis, vasodilation

2017

25. From the finding of hemoconcentration in localized venules during acute inflammation, one can directly infer that:
(A) Erythrocytes are escaping into the interstitial tissue.
(B) Leukocytes have successfully phagocytosed the invading pathogen.
(C) Intravascular fluid has been lost to the extravascular space.
(D) The local tissue has undergone coagulative necrosis.

2004

26. Which of the following represents an advantage of endothelial transcytosis during acute inflammation?
(A) It facilitates transport of specific proteins across intact endothelial cells.
(B) It allows rapid, massive fluid leakage that mimics the effects of direct endothelial cell necrosis.
(C) It completely prevents all plasma proteins from exiting the vessel, conserving intravascular osmotic pressure.
(D) It completely halts leukocyte diapedesis within inflamed venules to prevent secondary tissue damage.

2002

27. What is the definition of endothelial cell contraction in the context of vascular permeability?
(A) The irreversible detachment of damaged endothelial cells from the underlying basement membrane
(B) The apoptotic shrinkage of endothelial cells caused by direct toxic injury from bacterial endotoxins
(C) The rapid proliferation of vascular endothelial cells that physically crowds the microvascular lumen
(D) The reversible widening of intercellular junctions in post-capillary venules

2014

28. Which mediator is predominantly responsible for eliciting the rapid, reversible endothelial cell contraction seen in the immediate transient response?
(A) Transforming growth factor-beta
(B) Histamine
(C) Interleukin-10
(D) Vascular endothelial growth factor

2025

29. Identify the correct statement regarding direct endothelial injury.
(A) It occurs exclusively in post-capillary venules and never in capillaries or arterioles.
(B) It is typically mediated entirely by vasoactive amines such as histamine and bradykinin.
(C) It results in an immediate sustained response lasting until vessels are repaired.
(D) It causes an increase in permeability that peaks within 15 minutes and resolves within 30 minutes of injury.

NODIA

2013

30. Consider the following statements regarding the mechanisms that lead to increased vascular permeability in acute inflammation:
I. Endothelial contraction is mediated by histamine.
II. Endothelial transcytosis is mediated by vascular endothelial growth factor.
III. Endothelial proliferation is mediated by transforming growth factor-beta.
Which of the above statement(s) is/are correct?
(A) I
(B) II and III
(C) I and II
(D) III

2010

31. What is the direct effect of leukocyte-mediated endothelial injury during the inflammatory response?
(A) Toxic oxygen species and proteolytic enzymes from leukocytes damage the endothelium.
(B) Leukocytes physically block the post-capillary venules, causing an ischemic burst of the vessel wall.
(C) Leukocytes secrete abundant fibrinogen, which physically tears the tight endothelial junctions apart.
(D) Leukocytes downregulate selectin expression, forcing the vascular vessel to become highly permeable to compensate.

2003

32. In a burn patient, there is severe, widespread blistering with massive fluid loss. This fluid shift is primarily the application of which mechanism of vascular permeability?

(A) Endothelial contraction
(B) Direct endothelial injury
(C) Leukocyte-mediated endothelial injury
(D) Increased transcytosis

2024
33. Under which classification does the delayed prolonged response of vascular permeability typically fall?
(A) It is primarily an immediate histamine-mediated response characterized by rapid onset and very short duration.
(B) It is associated with mild thermal injury, radiation, or certain bacterial toxins.
(C) It is exclusively a result of rapid endothelial cell apoptosis triggered by direct mechanical trauma.
(D) It is defined by rapid resolution within approximately 30 minutes of the initiating traumatic injury.

2004
34. When comparing an exudate to a transudate, which specific gravity is most characteristic of an exudate?
(A) Greater than 1.020
(B) Less than 1.012
(C) Exactly 1.015
(D) Less than 1.005

2009
35. All of the following are biochemical or cellular characteristics of an inflammatory exudate, EXCEPT:
(A) High protein content (usually > 3 g/dL)
(B) Abundant presence of inflammatory cells
(C) Low specific gravity (< 1.012)
(D) Elevated lactate dehydrogenase levels

2009
36. Which of the following provides the most appropriate clinical criterion to differentiate an exudative pleural effusion from a transudative one?
(A) The fluid is perfectly clear and colorless.
(B) The fluid contains predominantly red blood cells rather than white blood cells.
(C) The fluid resolves spontaneously upon administration of diuretics.
(D) The ratio of pleural fluid protein to serum protein is greater than 0.5.

2002
37. From the presence of a transudate in the peritoneal cavity (ascites), what can be clinically inferred about the underlying pathophysiology?
(A) There is severe acute localized peritonitis with active recruitment of neutrophils.
(B) The vascular endothelium has undergone profound structural damage throughout the abdomen.
(C) There is hydrostatic or osmotic imbalance with intact endothelial barriers.
(D) There is widespread, active bacterial infection within the peritoneal abdominal cavity.

2024
38. What is the physiological advantage of forming a protein-rich exudate at the site of tissue injury?
(A) It completely prevents the migration of macrophages to avoid tissue destruction.
(B) It dilutes toxins and delivers immunoglobulins and fibrinogen to the damaged area.
(C) It lowers the local tissue oncotic pressure to pull fluid back into the blood vessels.
(D) It prevents the coagulation cascade from activating locally.

2009
39. What is the exact definition of a transudate?
(A) A protein-rich fluid with high cellular debris that directly results from increased vascular permeability.
(B) A localized, enclosed collection of purulent exudate caused by the invasion of pyogenic bacteria.
(C) A serous-type fluid containing large amounts of highly polymerized fibrin and cellular breakdown products.
(D) An ultrafiltrate of blood plasma with low protein content from hydrostatic or osmotic imbalance.

2006
40. Consider the following properties associated with inflammatory effusions:
I. Exudate: High protein content
II. Transudate: High specific gravity (> 1.020)
III. Exudate: Presence of cellular debris
IV. Transudate: Caused by normal vascular permeability
Which of the above statement(s) is/are incorrect?
(A) I, II and III
(B) II
(C) I and IV
(D) III and IV

2006
41. Identify the correct statement about the cellular composition of exudates.

Click Here to Buy book on Amazon

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

(A) Purulent exudates are entirely and consistently devoid of active polymorphonuclear neutrophils.
(B) Transudates typically contain a much higher concentration of leukocytes than inflammatory exudates.
(C) Exudates often contain polymorphonuclear leukocytes during acute phases.
(D) Monocytes are the exclusive and primary cell type found in any acute inflammatory tissue exudate.

2025
42. Identify the INCORRECT statement regarding the physical differences between exudates and transudates.
(A) Transudates contain high levels of fibrinogen and clot spontaneously.
(B) Exudates are frequently turbid, cloudy, or completely opaque in appearance.
(C) Transudates do not typically coagulate or form fibrin clots upon standing.
(D) Exudates may be classified as purulent, hemorrhagic, or highly fibrinous.

2012
43. What is the direct physiological effect of the expression of P-selectin and E-selectin on activated endothelial cells?
(A) Firm and stable adhesion of circulating leukocytes to the activated endothelium
(B) Active transmigration and diapedesis of leukocytes across the vascular basement membrane
(C) Immediate, explosive degranulation of marginated neutrophils in the vascular lumen
(D) Leukocyte rolling and transient attachment along the endothelial surface

2017
44. In a clinical condition like Leukocyte Adhesion Deficiency type 1 (LAD-1), patients lack functional beta-2 integrins (e.g., LFA-1, Mac-1). Which step of the leukocyte adhesion cascade is directly impaired?
(A) Margination
(B) Integrin-mediated firm adhesion
(C) Selectin-mediated rolling
(D) Chemotaxis through the interstitial tissue

2017
45. Which of the following adhesion molecules is classified as an integrin ligand belonging to the immunoglobulin superfamily?
(A) L-selectin
(B) Sialyl-Lewis X
(C) ICAM-1
(D) CD31 (PECAM-1)

2025
46. Comparing the roles of different selectins, which one is primarily expressed on the surface of the leukocytes rather than the endothelium?
(A) E-selectin
(B) P-selectin
(C) M-selectin
(D) L-selectin

NODIA

2008
47. All of the following molecules are essential for the firm adhesion phase of the leukocyte cascade, EXCEPT:
(A) Sialyl-Lewis X
(B) LFA-1 integrin
(C) Mac-1 integrin
(D) VCAM-1 adhesion molecule

2003
48. Which of the following is the most appropriate description of diapedesis?
(A) The passive diffusion of red blood cells through widened intercellular endothelial junctions
(B) Leukocyte transmigration across the endothelial layer and basement membrane
(C) The peripheral positioning of activated leukocytes along the endothelial wall during stasis
(D) The rapid phagocytosis of apoptotic endothelial cells by tissue-resident macrophages

2002
49. From the observation that neutrophils rapidly express high-affinity integrins after encountering chemokines on the endothelium, what can be inferred about the nature of integrin activation?
(A) Integrin binding affinity is a static physical property fixed during initial cell maturation in bone marrow.
(B) Chemokines induce a conformational change in integrins to a high-affinity state.
(C) Integrins must be newly synthesized from genomic DNA after the neutrophil first contacts the endothelium.
(D) Integrin activation within the microvasculature relies strictly on the presence of histamine in the blood.

2022

50. Consider the following molecules and their proposed primary roles in the leukocyte adhesion cascade:

I. P-selectin: Rolling
II. ICAM-1: Firm Adhesion
III. PECAM-1 (CD31): Transmigration
IV. Sialyl-Lewis X: Firm Adhesion
V. Integrins: Transmigration

Which of the above statement(s) is/are correct?

(A) I, II and III
(B) II, IV and V
(C) I, III and IV
(D) I, III, IV and V

2022

51. What is the formal definition of chemotaxis in the context of inflammation?

(A) The random, non-directional movement and tumbling of leukocytes within the bloodstream
(B) The cellular process by which phagocytic leukocytes ingest and destroy foreign particles
(C) Leukocyte migration along a chemical gradient toward the site of injury
(D) The localized synthesis and secretion of chemokines by tissue-resident macrophages

2003

52. Which of the following endogenous molecules acts as a potent chemoattractant for neutrophils?

(A) Interleukin-10
(B) Bradykinin
(C) Prostacyclin (PGI_2)
(D) Leukotriene B_4

2015

53. Identify the correct statement regarding the mechanism of leukocyte locomotion during chemotaxis.

(A) Leukocytes move primarily by extending long, specialized flagella from their posterior ends.
(B) Chemotaxis occurs through a passive mechanism that does not require intracellular calcium mobilization.
(C) Locomotion is driven by actin polymerization at the leading edge of the cell.
(D) Leukocytes travel against the concentration gradient of the target chemoattractant in the tissue.

2003

54. Identify the INCORRECT statement regarding chemoattractants.

(A) Bacterial products with N-formylmethionine terminal amino acids are potent exogenous chemoattractants.
(B) Interleukin-8 (CXCL8) selectively attracts eosinophils during allergic responses.
(C) C5a is a major endogenous chemoattractant derived from the complement system.
(D) Chemokines bind to specific seven-transmembrane G-protein-coupled receptors on leukocytes.

2004

55. What is the downstream cellular effect when a chemoattractant binds to a G-protein-coupled receptor on a leukocyte?

(A) Immediate activation of apoptosis within the leukocyte to prevent excessive tissue inflammation
(B) Direct downregulation of surface integrin affinity to facilitate rapid departure from the wall
(C) Comprehensive suppression of the metabolic oxidative burst mechanism during early phagocytosis
(D) Activation of phospholipase C and increased calcium, causing cytoskeletal changes

2010

56. A patient is infected with a bacteria that completely degrades C5a. How will this specifically affect the acute inflammatory response?

(A) Neutrophil migration to the infection site will be impaired.
(B) Neutrophil rolling along the vascular endothelium will be completely and permanently abolished.
(C) Downstream vasodilation mediated directly by histamine release will be prevented.
(D) Fibrin meshwork formation in the extravascular space will be excessively enhanced.

2024

57. How are chemokines structurally classified into subfamilies (e.g., CXC, CC)?

(A) Based on their absolute molecular weight measured in kilodaltons
(B) Based on the specific target inflammatory cell they act upon in tissues
(C) Based on the arrangement of conserved cysteine residues
(D) Based on their functional ability to activate the classical complement cascade

Downloaded from www.pdf.tube

2005

58. Comparing exogenous and endogenous chemoattractants, which of the following pairs correctly represents one of each?
(A) Exogenous: N-formylmethionyl peptides; Endogenous: Leukotriene B_4
(B) Exogenous: C5a; Endogenous: Interleukin-8
(C) Exogenous: Arachidonic acid; Endogenous: Lipopolysaccharide
(D) Exogenous: Fibrin degradation products; Endogenous: Peptidoglycan

2002

59. Which of the following is NOT a recognized major stage of the phagocytic process?
(A) Opsonization of the particle and subsequent receptor recognition
(B) Pseudopod engulfment and formation of the early phagosome
(C) Intracellular phagolysosome fusion
(D) Extracellular release of lysosomes

2003

60. Select the correct sequence of events during phagocytosis.
(A) Engulfment → Opsonization → Phagolysosome formation → Microbial killing
(B) Opsonization → Engulfment → Phagolysosome formation → Microbial killing
(C) Phagolysosome formation → Engulfment → Microbial killing → Opsonization
(D) Engulfment → Phagolysosome formation → Opsonization → Microbial killing

2011

61. From the requirement of actin polymerization for pseudopod extension during phagocytosis, what can be inferred about the effect of cytochalasin (an actin inhibitor) on macrophages?
(A) It will actively enhance the rate of the pseudopod engulfment phase.
(B) It will exclusively block the metabolic oxygen-dependent respiratory burst.
(C) It will prevent engulfment of opsonized particles.
(D) It will significantly increase the rate of intracellular phagolysosome fusion.

2012

62. What is the functional advantage of opsonization in the phagocytic process?
(A) It drastically reduces the total amount of metabolic energy required by the macrophage for chemotaxis.
(B) It causes the direct, antibody-mediated osmotic lysis of the invading microbe before engulfment occurs.
(C) It completely prevents the accidental, non-specific phagocytosis of surrounding healthy host cells.
(D) It coats microbes with molecules recognized by leukocyte receptors to enhance attachment.

2005

63. What is the definition of a phagosome?
(A) An intracellular vacuole containing an engulfed particle
(B) An extracellular vesicle released by neutrophils to trap bacteria
(C) A lysosome that has exhausted its enzymatic contents
(D) A nuclear structure responsible for synthesizing antimicrobial peptides

2001

64. Which two molecules are considered the major naturally occurring opsonins in host defense?
(A) Vasoactive histamine and serotonin
(B) IgG antibodies and the C3b fragment
(C) Pro-inflammatory leukotriene B_4 and prostaglandin E_2
(D) Cytokines interleukin-1 and tumor necrosis factor

2004

65. Identify the correct statement about the engulfment phase of phagocytosis.
(A) Engulfment is a passive process requiring no cellular ATP or metabolic energy expenditure.
(B) The cell membrane zippers around the particle to form an enclosed vacuole.
(C) Engulfment can only occur if the target microbe is classified strictly as a virus.
(D) The engulfment phase is mediated entirely by microtubules, with absolutely no actin involvement.

NODIA

2004

66. Identify the INCORRECT statement regarding the receptors involved in phagocytosis.
(A) Fc receptors on phagocytes bind to the Fab portion of IgM antibodies to initiate engulfment.
(B) Macrophage mannose receptors bind specifically to terminal mannose and fucose residues of microbial glycoproteins.
(C) Scavenger receptors bind to modified low-

density lipoproteins and various microbes.
(D) CR1 is a receptor on phagocytes that specifically recognizes the C3b opsonin.

2008

67. What is the immediate effect of the fusion between a phagosome and a lysosome?
(A) The immediate, active release of the engulfed particle back into the surrounding extracellular space
(B) The rapid, de novo synthesis of new mitochondrial DNA within the membrane-bound vacuole
(C) The formation of a phagolysosome, exposing the particle to hydrolytic enzymes
(D) The rapid, direct conversion of the functional lysosome into a protective peroxisome

2023

68. In chronic granulomatous disease, patients have a genetic defect in the phagocyte oxidase enzyme complex. Which mechanism of intracellular killing is primarily applied or affected here?
(A) The intracellular synthesis of nitric oxide by inducible nitric oxide synthase enzyme
(B) The enzymatic degradation of peptidoglycan cell walls by lysosomal lysozyme
(C) The biochemical sequestering of free environmental iron by lactoferrin
(D) The generation of superoxide anion via the respiratory burst

2019

69. How are intracellular microbial killing mechanisms broadly classified?
(A) Histamine-mediated and serotonin-mediated mechanisms
(B) Coagulation-dependent and kinin-dependent mechanisms
(C) Oxygen-dependent and oxygen-independent mechanisms
(D) Cytoplasmic and nuclear mechanisms

2011

70. Arrange the sequence of molecules generated during the oxygen-dependent respiratory burst, then select the matching option.
1. Superoxide anion
2. Hydrogen peroxide
3. Hypochlorous acid
4. Molecular oxygen

(A) 4, 1, 2, 3
(B) 1, 2, 4, 3
(C) 4, 2, 1, 3
(D) 2, 1, 3, 4

2001

71. Which of the following is NOT a component of the oxygen-independent intracellular killing mechanism?
(A) Lysozyme
(B) Major basic protein
(C) Defensins
(D) Myeloperoxidase

2017

72. Which is the most potent microbicidal system within neutrophils?
(A) The H_2O_2-myeloperoxidase-halide system
(B) The oxygen-independent lysozyme-lactoferrin system
(C) The eosinophil-derived major basic protein system
(D) The lysosomal acid hydrolase enzyme system

2003

73. From the presence of inducible nitric oxide synthase (iNOS) in activated macrophages, what can be inferred about its role in microbial killing?
(A) iNOS directly converts intracellular hydrogen peroxide into neutral water and molecular oxygen.
(B) iNOS actively prevents the physical fusion of acidic lysosomes with target phagosomes.
(C) iNOS produces nitric oxide, which reacts with superoxide to form toxic peroxynitrite.
(D) iNOS strictly functions to regulate localized blood vessel dilation around the tissue macrophage.

2008

74. What is a specific advantage of the oxygen-independent killing mechanisms?
(A) They can function in hypoxic environments where the respiratory burst fails.
(B) They produce vast and highly concentrated amounts of bactericidal hypochlorous acid.
(C) They represent the exclusive biochemical method by which intracellular viruses are destroyed.
(D) They do not require the formation of an enclosed cytoplasmic phagolysosome to function.

2014

75. What is the definition of the "respiratory burst" in the context of phagocytes?
(A) A sudden, rapid exhalation of carbon dioxide by the cell during acute metabolic stress
(B) The destruction of functional mitochondrial respiration by specialized bacterial toxins
(C) The explosive, receptor-mediated

Click Here to Buy book on Flipkart

degranulation of tissue-resident mast cells
(D) A transient increase in oxygen consumption during phagocytosis to produce ROS

2013
76. During an acute pyogenic bacterial infection, which cell type exhibits the most rapid kinetics, typically dominating the inflammatory infiltrate within the first 6 to 24 hours?
(A) Lymphocytes
(B) Neutrophils
(C) Macrophages
(D) Eosinophils

2015
77. Identify the correct statement concerning the morphology of eosinophils.
(A) They possess a single, perfectly round nucleus and a completely clear, agranular cytoplasm.
(B) They are the largest leukocytes, characterized by a kidney-bean shaped nucleus and clear cytoplasm.
(C) They contain bi-lobed nuclei and large, intensely acidophilic granules.
(D) They completely lack any distinct, stainable cytoplasmic granules under routine light microscopy.

2014
78. Identify the INCORRECT statement regarding mast cells.
(A) They are widely distributed in connective tissues adjacent to blood vessels.
(B) They store preformed histamine in their metachromatic granules.
(C) They degranulate upon binding of specific antigens to IgE attached to their surface receptors.
(D) They are strictly found in the circulation and never reside in solid tissues.

2007
79. What is the primary cause of the shift from a neutrophil-predominant infiltrate to a macrophage-predominant infiltrate after 24-48 hours in acute inflammation?
(A) Neutrophils undergo rapid apoptosis in tissue, whereas macrophages survive much longer.
(B) Neutrophils directly differentiate and replicate into functional tissue macrophages after 24 hours.
(C) Macrophages migrate much faster than neutrophils in response to chemokines but take longer to differentiate.
(D) The local microvascular blood supply completely shuts off, selectively killing all tissue neutrophils.

2004
80. Assess the following assertion and reason regarding eosinophils, and choose the correct option.
Assertion (A): Eosinophils are prominently featured in inflammatory reactions mediated by IgE and in parasitic infections.
Reason (R): Eosinophil granules contain major basic protein, which is highly toxic to helminth parasites but cannot phagocytose regular bacteria.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2015
81. How are basophils primarily classified in terms of their lineage and structural characteristics?
(A) Agranular mononuclear phagocytes that migrate into tissues to become macrophages
(B) Granulocytes with basic-staining granules rich in histamine
(C) Tissue-fixed helper T lymphocytes responsible for mediating delayed hypersensitivity
(D) Anucleated, membrane-bound cellular fragments derived directly from megakaryocytes

2004
82. When comparing mast cells and basophils, which feature accurately distinguishes their typical physiological localization?
(A) Mast cells reside in tissues, while basophils circulate in blood.
(B) Both cells function exclusively as circulating leukocytes within the vascular system.
(C) Basophils reside in tissues, while mast cells circulate in blood.
(D) Both cells are found exclusively in the bone marrow as undifferentiated stem cells.

2012
83. All of the following are specific functions of neutrophils in acute inflammation, EXCEPT:
(A) Active phagocytosis and intracellular destruction of opsonized bacteria
(B) Production of toxic reactive oxygen species

Downloaded from www.pdf.tube

via the metabolic respiratory burst
(C) Presentation of processed antigens to naive T-cells
(D) Neutrophil extracellular trap (NET) formation to physically ensnare pathogens

2009

84. Which characteristic is most appropriate for describing the nuclear morphology of a mature, functioning neutrophil?
(A) A large, monolithic nucleus occupying approximately 90% of the total cellular volume
(B) A bi-lobed nucleus presenting with a highly characteristic and symmetric "spectacle" appearance
(C) A completely fragmented, non-functional, and physically extruded nucleus from the cytoplasm
(D) A multi-lobulated nucleus with 3 to 5 lobes connected by fine chromatin

2004

85. From the observation that antihistamine medications rapidly reduce the wheal and flare response in allergic skin tests, what can be inferred about the timing of histamine action?
(A) It requires active de novo synthesis within the nucleus, taking several hours to complete.
(B) It acts exclusively in the late, chronic phases of inflammation rather than acute reactions.
(C) It is preformed and released immediately upon mast cell stimulation.
(D) It relies entirely on the upstream activation of the classical complement cascade to function.

2008

86. What is a key physiological advantage of storing vasoactive amines in preformed cytoplasmic granules?
(A) It completely prevents stored vasoactive amines from ever undergoing enzymatic degradation inside the host cell.
(B) It allows immediate release of mediators without the delay of transcription.
(C) It ensures that the released amines only act in an autocrine fashion on the exact cell that produced them.
(D) It directly and selectively enhances the mitochondrial production of reactive oxygen species during activation.

NODIA

2024

87. What defines a "vasoactive amine" in the context of inflammation?
(A) Any specialized protein derived from plasma that actively modulates systemic systemic blood pressure.
(B) A lipid-derived, newly synthesized mediator that strictly causes localized arteriolar vasoconstriction.
(C) Active, secreted enzyme that selectively breaks down the surrounding extracellular matrix proteins.
(D) A nitrogenous compound that induces vascular caliber and permeability changes.

2021

88. Which cell types serve as the primary storage sites for serotonin (5-hydroxytryptamine) during the inflammatory response?
(A) Platelets and enterochromaffin cells
(B) Circulating neutrophils and tissue macrophages
(C) Vascular endothelial cells and dermal fibroblasts
(D) Helper T-lymphocytes and antibody-producing B-lymphocytes

2022

89. Which of the following is the most potent physiological stimulus for histamine release from tissue mast cells during an acute inflammatory response?
(A) Binding of antigen to IgE already attached to FcεRI receptors on mast cells
(B) Activation of cyclooxygenase by arachidonic acid
(C) Direct stimulation by prostaglandin E_2
(D) Binding of C3b to complement receptors on mast cells

2003

90. Match the vasoactive amines or characteristics in List I with their corresponding associations in List II:

List I: Vasoactive Amine / Characteristic
P. Histamine primary source
Q. Serotonin primary source
R. Histamine vascular effect
S. Serotonin vascular effect (in inflammation)

List II: Association
1. Arteriolar vasodilation & venular permeability
2. Vasoconstriction (in some beds) or vasodilation/permeability
3. Platelets

4. Mast cells
(A) P-4, Q-3, R-1, S-2
(B) P-3, Q-4, R-2, S-1
(C) P-3, Q-1, R-4, S-2
(D) P-4, Q-3, R-2, S-1

2009

91. What is the direct effect of when histamine binds to H1 receptors on post-capillary venule endothelial cells?
(A) Localized endothelial cell proliferation and angiogenesis
(B) Rapid intracellular synthesis of extracellular collagen type I
(C) Irreversible, programmed apoptosis of the vascular endothelium
(D) Endothelial cell contraction and increased permeability

2004

92. A patient with severe allergies is administered an inhibitor of mast cell degranulation. Which stage of acute inflammation will this clinically apply to and directly dampen the most?
(A) The delayed prolonged response of permeability
(B) The immediate transient response
(C) The fibroblast-mediated synthesis of scar tissue
(D) The systemic, TNF-mediated acute-phase fever response

2012

93. Based on their synthetic pathways, how are arachidonic acid metabolites (eicosanoids) broadly classified?
(A) Endocrine hormones and localized paracrine cytokines
(B) Preformed vasoactive amines and newly synthesized proteins
(C) Cyclooxygenase and lipoxygenase products
(D) Classical pathway components and alternative pathway factors

2024

94. Comparing prostaglandins and leukotrienes, which statement correctly differentiates their primary physiological roles in acute inflammation?
(A) Prostaglandins primarily mediate targeted neutrophil chemotaxis, while leukotrienes primarily mediate central fever.
(B) Prostaglandins are synthesized via the lipoxygenase pathway, while leukotrienes are synthesized by cyclooxygenase.
(C) Prostaglandins consistently inhibit localized inflammation, while leukotrienes exclusively act to initiate it.
(D) Prostaglandins mediate vasodilation/pain, while leukotrienes mediate chemotaxis/permeability.

2014

95. All of the following are products of the cyclooxygenase pathway, EXCEPT:
(A) Leukotriene B_4 (LTB_4)
(B) Prostagandin E_2 (PGE_2)
(C) Prostacyclin (PGI_2)
(D) Thromboxane A_2 (TXA_2)

2010

96. Which of the following arachidonic acid metabolites is most appropriately identified as a potent inhibitor of platelet aggregation and a powerful vasodilator?
(A) Thromboxane A_2
(B) Prostacyclin (PGI_2)
(C) Leukotriene C_4
(D) Prostaglandin F_2-α

2024

97. If a nonsteroidal anti-inflammatory drug (NSAID) specifically blocks the cyclooxygenase enzyme, what can be inferred about the levels of arachidonic acid metabolites?
(A) Both localized prostaglandin and leukotriene synthesis pathways will be completely and rapidly halted.
(B) Prostaglandin/thromboxane levels will drop, but leukotriene production may continue.
(C) Only the specialized, resolution-promoting lipoxin synthesis pathway will be selectively inhibited.
(D) The biochemical release of arachidonic acid from membrane phospholipids will be completely prevented.

2005

98. What is the primary physiological advantage of synthesizing lipoxins during an inflammatory response?
(A) They act as negative regulators that promote resolution by inhibiting neutrophil recruitment.
(B) They physically and irreversibly bind to and destroy the structural integrity of bacterial cell walls.
(C) They severely constrict localized microvascular blood vessels to prevent hemorrhage and fluid loss.

Click Here to Buy book on Amazon

(D) They drastically amplify the downstream cellular production of highly pro-inflammatory cytokines like TNF.

2025

99. Which statement defines the origin of arachidonic acid used in the production of eicosanoids?

(A) It is a circulating plasma protein synthesized and continuously secreted into blood by the liver.

(B) It is a preformed vasoactive amine stored in the cytoplasmic metachromatic granules of mast cells.

(C) It is a 20-carbon fatty acid released from cell membrane phospholipids by phospholipase A_2.

(D) It is an exogenous chemical compound absorbed exclusively and directly from bacterial outer membranes.

2009

100. Consider the following statements regarding arachidonic acid metabolites and their physiological effects:

I. Thromboxane A_2: Vasoconstriction and platelet aggregation

II. Leukotriene B_4: Inhibitor of neutrophil chemotaxis

III. Prostaglandin E_2: Mediates pain and fever

Which of the above statement(s) is/are correct?

(A) I

(B) II and III

(C) I and III

(D) I and II

2006

101. Identify the correct statement regarding the complement cascade in acute inflammation.

(A) The alternative pathway is activated exclusively by preformed circulating antigen-antibody complexes.

(B) The membrane attack complex (MAC) is composed primarily of early complement factors C1, C2, and C_4.

(C) C3a and C5a act as anaphylatoxins, stimulating histamine release.

(D) C3b actively inhibits the process of phagocytosis by completely blocking leukocyte surface receptors.

2012

102. Identify the INCORRECT statement regarding the kinin system.

(A) Activation of the kinin system leads to the formation of bradykinin.

(B) Bradykinin is a potent long-acting vasoconstrictor.

(C) Bradykinin causes increased vascular permeability and pain.

(D) The kinin system is initiated by the activation of Hageman factor (Factor XII).

2007

103. What is the direct effect of Hageman factor (Factor XII) activation on the interplay of plasma protein-derived mediators?

(A) It exclusively triggers the classical complement pathway without affecting other cascades.

(B) It rapidly deactivates the complement membrane attack complex to protect surrounding host cells.

(C) It causes the immediate, rapid enzymatic degradation of bradykinin within the local tissue.

(D) It triggers coagulation, kinin, and fibrinolytic systems simultaneously.

2015

104. In a clinical scenario of disseminated intravascular coagulation (DIC), widespread activation of the coagulation cascade occurs. Which system acts to limit this clotting by cleaving fibrin, producing split products that increase vascular permeability?

(A) The fibrinolytic system

(B) The plasma-derived kinin system

(C) The multi-protein complement system

(D) The metabolic cyclooxygenase pathway

2025

105. How are the plasma protein-derived mediators broadly classified based on their activation mechanism?

(A) They are synthesized on demand by local tissue macrophages in response to cellular damage.

(B) They are preformed inside blood platelets and rapidly released upon mechanical degranulation.

(C) They circulate as inactive precursors requiring proteolytic cleavage.

(D) They are exclusively derived from arachidonic acid metabolism in response to membrane injury.

2013

106. Comparing the vascular effects of bradykinin and histamine, which of the following is true?

(A) Histamine is a long-acting peptide, whereas

NODIA

bradykinin is a short-acting amine.
(B) Bradykinin induces pain, whereas histamine generally does not.
(C) Bradykinin causes vasoconstriction, whereas histamine causes vasodilation.
(D) Histamine requires proteolytic cleavage from plasma precursors, whereas bradykinin is stored in granules.

2003

107. All of the following are recognized roles of plasmin within the inflammatory interplay, EXCEPT:
(A) Synthesizing prostaglandins from arachidonic acid
(B) Cleaving insoluble fibrin to yield fibrin degradation products
(C) Cleaving complement protein C3 to generate active C3 fragments
(D) Activating Hageman factor (Factor XII) to amplify the pathway

2004

108. Which component of the complement cascade is most appropriately recognized as the critical convergence point for the classical, alternative, and lectin pathways?
(A) C1q
(B) C5
(C) C3
(D) C9

2024

109. From the action of C5a in acute inflammation, one can infer that its presence in tissue will lead to:
(A) Immediate suppression of the respiratory burst in local macrophages.
(B) Irreversible inhibition of the active lipoxygenase pathway in leukocytes.
(C) Prompt formation of a stable fibrin clot within the vascular lumen.
(D) Recruitment of neutrophils via chemotaxis and increased permeability.

2002

110. Consider the following statements regarding pro-inflammatory cytokines and their roles:
I. TNF: Promotes endothelial expression of selectins
II. IL-1: Strongly inhibits systemic fever
III. IL-6: Stimulates hepatic synthesis of acute-phase proteins
IV. IL-10: Potent stimulator of acute inflammation
Which of the above statements are INCORRECT?
(A) I, II and III
(B) II and IV
(C) I and III
(D) III and IV

2017

111. What is the definition of a cytokine in the context of the immune system?
(A) A lipid-derived mediator responsible primarily for local pain modulation and signaling.
(B) A small secreted protein regulating host immune and inflammatory responses.
(C) A large plasma glycoprotein forming the primary structural framework of a blood clot.
(D) An intracellular organelle responsible for digesting Gulfed pathogenic microbes.

2014

112. Which two cytokines are recognized as the primary mediators of the acute-phase response, including fever and the systemic release of acute-phase proteins?
(A) Interleukin-4 and Interleukin-5
(B) Transforming Growth Factor-beta (TGF-β) and Interleukin-10
(C) Interferon-gamma and Interleukin-2
(D) Tumor Necrosis Factor (TNF) and Interleukin-1 (IL-1)

2010

113. Identify the correct statement regarding the local effects of Tumor Necrosis Factor (TNF).
(A) TNF induces endothelial activation, promoting leukocyte adhesion and coagulation.
(B) TNF strictly downregulates the expression of adhesion molecules on endothelial cells.
(C) TNF limits the inflammatory response by inducing rapid apoptosis of all local macrophages.
(D) TNF is synthesized primarily by eosinophils during chronic parasitic infections.

2003

114. Identify the INCORRECT statement regarding the systemic effects of acute inflammation mediated by cytokines.
(A) IL-1 and TNF induce systemic fever by acting directly on the hypothalamus to upregulate prostaglandins.
(B) IL-6 stimulates hepatocyte protein synthesis, increasing systemic C-reactive protein and fibrinogen levels.

Downloaded from www.pdf.tube

(C) TNF limits bone marrow release, causing severe chronic leukopenia as a primary acute phase response.
(D) Elevated systemic levels of TNF in severe infections can lead to massive vasodilation and septic shock.

2008

115. What is the direct cause of the accelerated erythrocyte sedimentation rate (ESR) seen during systemic acute inflammation?
(A) Fibrinogen coating red blood cells, causing rouleaux formation
(B) Increased blood viscosity due to acute hypernatremia and dehydration
(C) Massive destruction of red blood cells by splenic macrophages
(D) Complete systemic exhaustion of the circulating complement cascade

2001

116. A patient is administered a biologic drug that specifically neutralizes TNF. In the context of rheumatoid arthritis, how will this therapeutically apply to the inflamed joints?
(A) It will drastically increase the recruitment of active inflammatory leukocytes into the joint space.
(B) It will promote rapid periarticular fibroblast proliferation and irreversible joint ankylosis.
(C) It will directly dissolve the pre-existing chronic pannus tissue via direct enzymatic degradation.
(D) It will decrease endothelial activation, reducing leukocyte infiltration and joint damage.

2002

117. How are chemokines (e.g., IL-8) classified functionally compared to major pro-inflammatory cytokines like TNF and IL-1?
(A) Chemokines act primarily as systemic pyrogens, while TNF and IL-1 act exclusively as localized chemoattractants.
(B) Chemokines direct leukocyte migration, whereas TNF and IL-1 activate endothelium and systemic responses.
(C) Chemokines function as strictly inhibitory molecules, whereas TNF and IL-1 serve as broad systemic stimulatory proteins.
(D) Chemokines are classified as lipid-derived mediators, whereas TNF and IL-1 are structurally classified as proteins.

2008

118. Comparing serous and fibrinous inflammation, which characteristic best distinguishes fibrinous inflammation?
(A) It is characterized primarily by a watery, protein-poor transudate derived from the serum.
(B) It typically resolves within a few minutes without any possibility of subsequent tissue scarring.
(C) It involves high vascular permeability, letting fibrinogen escape and polymerize into fibrin.
(D) It is found localized exclusively in the mucosal linings and membranes of the respiratory tract.

2013

119. All of the following correctly describe purulent (suppurative) inflammation, EXCEPT:
(A) It is characterized by the production of large amounts of pus.
(B) It is commonly caused by infections with pyogenic bacteria, such as Staphylococcus.
(C) It contains prominent amounts of neutrophils, necrotic cells, and edema fluid.
(D) It primarily consists of a cell-free, low-protein ultrafiltrate.

NODIA

2003

120. Consider the following statements regarding morphological classifications of acute inflammation:
I. Serous: Watery, protein-poor fluid accumulation
II. Fibrinous: Extensive deposition of fibrin meshwork
III. Suppurative: Exudate rich in neutrophils and cell debris
IV. Catarrhal: Inflammation specifically involving mucin-producing epithelial surfaces
V. Hemorrhagic: Complete absence of red blood cells in the exudate
Which of the above statements are correct?
(A) I, II, III and V
(B) I, II, III and IV
(C) II, III, IV and V
(D) I, III, IV and V

2001

121. From the observation of a thick, opaque, yellow-green fluid completely filling an abscess cavity, one can accurately infer that the predominant inflammatory cell type present is:
(A) The eosinophil
(B) The neutrophil

(C) The lymphocyte
(D) The mature plasma cell

2004

122. What is the pathological disadvantage if fibrinous exudate in a body cavity (e.g., pericardium) is not promptly cleared by macrophages?
(A) It will transform over time into a permanent, highly aggressive malignant tumor of the mesothelium.
(B) It undergoes organization, causing fibroblast ingrowth and fibrous adhesions.
(C) It will cause rapid, immediate, and complete dystrophic calcification of the surrounding parenchymal organ.
(D) It will generate a state of continuous, uncontrolled, and life-threatening systemic anaphylaxis.

2008

123. What is the definition of catarrhal inflammation?
(A) Superficial inflammation of mucous membranes with excessive mucus secretion
(B) Inflammation characterized by profound vascular wall damage and massive local tissue hemorrhage.
(C) Inflammation resulting directly in the rapid liquefactive necrosis of cerebral brain tissue.
(D) The formation of an extensively ulcerated mucosal surface lined by necrotic caseous debris.

2023

124. Which morphological type of inflammation is historically best exemplified by the fluid inside a simple skin blister caused by a mild burn?
(A) Purulent inflammation
(B) Hemorrhagic inflammation
(C) Serous inflammation
(D) Fibrinous inflammation

2010

125. Identify the correct statement regarding hemorrhagic inflammation.
(A) It is clinically and histologically considered the mildest possible form of acute inflammation.
(B) It involves a highly specialized cellular exudate completely devoid of active leukocytes.
(C) It occurs primarily due to a chronic severe deficiency of dietary iron and other trace minerals.
(D) It implies vascular injury with erythrocyte leakage into tissue.

2008

126. Identify the INCORRECT statement regarding the morphological patterns of acute inflammation.
(A) Ulcers represent a local defect of the surface of an organ or tissue produced by the sloughing of inflamed necrotic tissue.
(B) Serous exudates can derive either from blood plasma or from the secretions of mesothelial cells lining pleural/peritoneal cavities.
(C) Suppurative inflammation inevitably heals by first intention without any scarring.
(D) Fibrinous inflammation is frequently observed on the linings of body cavities, such as the meninges, pericardium, and pleura.

2012

127. What is the fundamental cause of the shift from acute to chronic inflammation?
(A) The rapid, highly efficient, and successful phagocytosis of the primary offending agent.
(B) Persistent inciting stimuli or recurrent acute inflammatory episodes
(C) The complete and irreversible absence of blood supply to the deeply affected necrotic tissue.
(D) The sudden, massive, and localized release of highly active preformed vascular histamine.

2024

128. In a histological section of chronic inflammatory tissue, large numbers of plasma cells are observed. What function are these cells applying in this environment?
(A) They are actively and continuously phagocytosing dead or dying polymorphonuclear neutrophils.
(B) They are directly generating a dense extracellular collagen matrix to form a permanent fibrotic scar.
(C) They are laying down a thick insoluble fibrin meshwork to securely wall off the localized infection.
(D) They are secreting specific antibodies against persistent antigens.

2001

129. How are the predominant cellular players classified in chronic inflammation compared to acute inflammation?
(A) Chronic features mononuclear cells; acute features primarily polymorphonuclear cells.
(B) Chronic inflammation features exclusively neutrophils; acute inflammation features exclusively activated macrophages.
(C) Chronic inflammation features strictly tissue

Click Here to Buy book on Flipkart

eosinophils; acute inflammation features strictly mature lymphocytes.
(D) Chronic inflammation features lack cellular infiltrates entirely, relying solely on passive fluid exudation processes.

2002
130. Select the correct sequence of events that best illustrates the typical macrophage life cycle during chronic inflammation.
(A) Bone marrow stem cell → Blood monocyte → Tissue macrophage → Activation
(B) Blood monocyte → Bone marrow stem cell → Activation → Tissue macrophage
(C) Tissue macrophage → Blood monocyte → Activation → Bone marrow stem cell
(D) Activation → Bone marrow stem cell → Tissue macrophage → Blood monocyte

2012
131. All of the following are hallmark histological features of chronic inflammation, EXCEPT:
(A) Intense, diffuse neutrophilic infiltration and abundant pus formation
(B) Mononuclear cell infiltration
(C) Tissue destruction induced by persistent offending agents or inflammatory cells
(D) Attempts at healing by connective tissue replacement (angiogenesis and fibrosis)

2022
132. Which of the following describes the most appropriate bidirectional cross-talk mechanism between macrophages and lymphocytes in chronic inflammation?
(A) Lymphocytes secrete vasoactive histamine to directly activate macrophages; macrophages secrete functional antibodies to activate lymphocytes.
(B) Macrophages secrete soluble fibrinogen to coat target lymphocytes; lymphocytes secrete active plasmin to enzymatically free macrophages.
(C) Lymphocytes directly phagocytose heavily activated macrophages, presenting their cellular antigens to naïve circulating B-cells.
(D) Macrophages secrete IL-12 activating T-cells; T-cells secrete IFN-gamma activating macrophages.

2023
133. From the persistent presence of heavily activated macrophages in a tissue, what can be inferred about the local tissue environment?
(A) The local tissue is undergoing rapid, highly organized, complete and perfect regeneration back to its native anatomical state.
(B) The tissue suffers collateral damage from released reactive oxygen species, proteases, and cytokines.
(C) The tissue environment is completely sterile, functionally healthy, and entirely devoid of any foreign microbial antigens.
(D) The regional tissue vascularity will permanently and irreversibly shut down in order to physically isolate these macrophages.

NODIA

2010
134. What is the physiological advantage of macrophage accumulation during a prolonged infection?
(A) They are the sole immune cells capable of generating sudden, massive, and localized releases of preformed histamine.
(B) They rapidly and systematically undergo apoptosis at the site of injury to minimize collateral parenchymal damage.
(C) They provide continuous phagocytosis, antigen presentation, and repair signaling.
(D) They entirely and permanently bypass any biological requirement for initiating a subsequent adaptive immune response.

2017
135. What defines a granuloma?
(A) A highly localized collection of suppurative pus completely surrounded by a dense, protective fibrous connective tissue capsule.
(B) A highly malignant epithelial or mesenchymal tumor arising directly from the active granulation tissue of a healing wound.
(C) A widely diffuse, rapidly spreading necrotizing bacterial infection propagating through deep subcutaneous fascial planes.
(D) A focal accumulation of epithelioid macrophages, often surrounded by lymphocytes

2013
136. Which classical infectious agent is widely recalled as the prototypic cause of caseating granulomatous inflammation?
(A) Mycobacterium tuberculosis
(B) Staphylococcus aureus
(C) Streptococcus pyogenes
(D) Escherichia coli

2009
137. Identify the correct statement regarding epithelioid cells in a granuloma.

(A) They are terminally transformed polymorphonuclear neutrophils that have completely lost their lysosomal granules.
(B) Activated macrophages with abundant cytoplasm resembling epithelial cells
(C) They are true epidermal or mucosal epithelial cells that have actively migrated deep into the inflamed connective tissue.
(D) They possess highly specialized, massive flagella that allow them to migrate exceptionally fast throughout the granuloma.

2005

138. Identify the INCORRECT statement regarding multinucleated giant cells in granulomas.
(A) They are formed by the fusion of multiple activated macrophages.
(B) Multinucleated giant cells divide rapidly via mitosis to populate the granuloma.
(C) Langhans giant cells typically have their nuclei arranged in a horseshoe pattern at the cell periphery.
(D) Foreign body giant cells usually have nuclei haphazardly scattered throughout their cytoplasm.

2006

139. What is the primary immunologic cause of epithelioid macrophage transformation and giant cell formation in an immune granuloma?
(A) T-cell cytokine stimulation, particularly Interferon-gamma
(B) Unusually high levels of circulating systemic histamine released from mast cells.
(C) Direct, severe physical mechanical trauma to the outer macrophage cell membrane.
(D) Massive, sudden localized influx of the active circulating C3a anaphylatoxin.

2003

140. Arrange the typical components of a mature tuberculous granuloma from the innermost core to the outermost rim, then select the matching option.
1. Collar of lymphocytes
2. Caseous necrosis
3. Epithelioid macrophages and giant cells
4. Fibroblastic proliferation (fibrosis)

(A) 2, 3, 1, 4
(B) 1, 3, 4, 2
(C) 3, 2, 1, 4
(D) 2, 1, 3, 4

NODIA

2013

141. How are granulomas broadly classified based on their pathogenesis?
(A) Acute suppurative and localized serous reactions
(B) Hyperacute systemic and chronic localized types
(C) Diffuse fibrinous and superficial catarrhal lesions
(D) Immune and foreign body granulomas

2010

142. Comparing Langhans giant cells and Touton giant cells, which feature is highly characteristic of Touton giant cells?
(A) They strictly and consistently contain only two highly condensed nuclei resembling classical owl eyes.
(B) They are completely and structurally devoid of any cytoplasm, consisting solely of densely clustered nuclei.
(C) Peripheral ring of nuclei with foamy, lipid-rich cytoplasm
(D) They are specifically and uniquely derived from fused mature plasma cells rather than tissue macrophages.

2011

143. All of the following pathological conditions characteristically feature granulomatous inflammation, EXCEPT:
(A) Leprosy
(B) Acute appendicitis
(C) Syphilis
(D) Sarcoidosis

2010

144. Which classification most appropriately describes cells that continuously cycle to replace cells that are constantly being destroyed or shed?
(A) Permanent cells
(B) Quiescent cells
(C) Stable cells
(D) Labile cells

2016

145. From the classification of neurons and cardiac myocytes as "permanent" cells, what can be inferred about their response to severe injury?
(A) Being mitotic-incapable, destroyed cells are replaced by fibrous scar tissue.
(B) They will rapidly and continuously divide to perfectly and flawlessly restore the original tissue architecture.
(C) They will temporarily and completely dedifferentiate into multipotent stem cells

before undergoing final healing.
(D) They rely strictly and continuously on rapid superficial shedding mechanisms to maintain overall tissue health.

2005
146. What is the clinical advantage of the regenerative capacity of "stable" (quiescent) cells, such as hepatocytes?
(A) They constantly replicate on a daily basis, allowing for a continuous, highly rapid physiological turnover of the entire parenchymal organ.
(B) They never undergo any replication under any circumstance, ensuring that potential somatic genetic mutations are absolutely and completely prevented.
(C) They rest in G0 but enter the cell cycle to regenerate tissue after injury.
(D) They can easily and rapidly transdifferentiate into completely different embryonic germ layers to repair almost any damaged organ.

2023
147. What is the definition of stable (quiescent) cells in the context of tissue regeneration?
(A) Cells with low normal replication that divide in response to stimuli
(B) Cells that undergo continuous, highly rapid, and unchecked physiological division throughout the host's entire life.
(C) Cells that have left the cell cycle permanently, are terminally differentiated, and cannot undergo any further mitosis.
(D) Cells that exclusively and strictly divide during early embryonic development stages and never undergo division postnatally.

2007
148. Which of the following tissues is a classical example of a labile cell population?
(A) Differentiated skeletal muscle
(B) Renal tubular epithelium
(C) Central nervous system tissue
(D) Epidermal squamous skin cells

2004
149. Identify the correct statement regarding the limitations of tissue regeneration even in tissues with labile or stable cells.
(A) Regeneration can always occur flawlessly and rapidly, completely regardless of the overall extent of structural extracellular matrix damage.
(B) Stromal framework destruction leads to scarring rather than regeneration.
(C) Highly active labile cells are completely and naturally immune to all toxic chemical insults and never undergo pathological necrosis.
(D) Under physiological limits, stable cells are restricted and can only undergo regeneration exactly once in a host's entire lifetime.

2005
150. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option:
Assertion (A): Severe, extensive damage to the liver can result in cirrhosis (fibrotic scarring) despite hepatocytes being stable cells with high regenerative capacity.
Reason (R): The destruction of the reticulin framework in severe liver injury prevents replicating hepatocytes from organized structural regeneration, leading to nodule and scar formation.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2009
151. What causes permanent cells, such as adult cardiac myocytes, to heal via fibrosis rather than regeneration following a myocardial infarction?
(A) They lack the genetic machinery to synthesize collagen.
(B) They undergo extremely rapid mitosis that outpaces the blood supply, forming a dense scar.
(C) They are continuously shed into the bloodstream, depleting the local cell population.
(D) They are terminally differentiated and lack the capacity to enter the mitotic cell cycle.

2006
152. In a clinical scenario of a surgical incision, early application of sutures ensures the wound edges are approximated. Which stage of tissue repair does this intervention most directly facilitate by minimizing the tissue gap?
(A) Proliferation
(B) Remodeling
(C) Hemostasis
(D) Maturation

Downloaded from www.pdf.tube

2022

153. How are the stages of wound repair broadly classified in chronological order?

(A) Remodeling → Inflammation → Hemostasis → Proliferation

(B) Hemostasis → Inflammation → Proliferation → Remodeling

(C) Inflammation → Proliferation → Remodeling → Hemostasis

(D) Proliferation → Hemostasis → Inflammation → Remodeling

2013

154. Comparing the inflammation phase and the proliferation phase of tissue repair, which cellular activity heavily dominates the proliferation phase?

(A) Massive and continuous influx of neutrophils to clear local bacteria

(B) Angiogenesis and fibroblast collagen synthesis

(C) Intricate cross-linking of collagen to maximize final tensile strength

(D) Immediate and profound local vasoconstriction to rapidly halt bleeding

2019

155. All of the following are primary objectives of the hemostasis stage in tissue repair, EXCEPT:

(A) Extensive degradation of excess extracellular matrix

(B) Vasoconstriction to limit initial blood loss

(C) Formation of a platelet plug

(D) Activation of the coagulation cascade to form a stable fibrin clot

2003

156. Which of the following is the most appropriate description of the remodeling (maturation) phase of wound healing?

(A) It represents the shortest overall physiological phase, typically lasting only a few fleeting hours immediately after initial tissue injury.

(B) It is characterized primarily and pathologically by explosive, uncontrolled bacterial multiplication and acute suppurative pus formation.

(C) Replacing type III collagen with stronger type I and cross-linking

(D) It strictly and exclusively features the continuous, highly rapid proliferation of local endothelial cells to form new functional blood vessels.

2025

157. From the observation that a wound regains only about 70-80% of normal uninjured skin strength after several months, what can be inferred about scar tissue?

(A) Mature scar tissue represents a structurally and functionally perfect replication of the original uninjured native tissue.

(B) Fully formed scar tissue relies almost entirely on flexible elastin fibers rather than dense collagen for its tensile strength.

(C) The healing wound must undergo continuous, repetitive physical re-injury in order to finally gain 100% of its baseline strength.

(D) Remodeling improves tensile strength but never reaches pre-injury perfection.

2003

158. What is the advantage of early macrophage arrival during the inflammatory stage of repair?

(A) They produce and release massive systemic amounts of vasoactive histamine to physically keep the active wound edges widely open.

(B) They rapidly and directly transdifferentiate into active fibroblasts to completely and instantly seal the open wound site.

(C) They clear necrotic debris and secrete growth factors initiating the proliferative phase.

(D) They continuously and enzymatically dissolve the newly formed fibrin clot to absolute perfection, preventing any scarring.

NODIA

2016

159. What is the definition of the proliferative phase in the context of wound healing?

(A) The initial acute period characterized primarily by active fibrin clot formation and immediate, massive neutrophilic influx.

(B) Period characterized by granulation, epithelialization, and contraction

(C) The final long-term physiological period characterized primarily by profound, progressive scar blanching and avascularity.

(D) The early pathological period characterized primarily by widespread, irreversible ischemic cell death and tissue necrosis.

2016

160. Match the stage of tissue repair in List I with its hallmark event in List II:

List I: (Stage of tissue repair)

P. Hemostasis

Q. Inflammation

R. Proliferation

S. Remodeling

List II: (Hallmark event)

1. Granulation tissue formation
2. Fibrin clot formation
3. Neutrophil and macrophage infiltration
4. Collagen cross-linking and scar contraction

(A) P-2, Q-3, R-1, S-4
(B) P-3, Q-1, R-4, S-2
(C) P-1, Q-4, R-2, S-3
(D) P-2, Q-1, R-3, S-4

2012

161. Identify the correct statement regarding the macroscopic appearance of healthy granulation tissue.
(A) Pink-to-red, soft, granular, bleeding easily upon minor trauma.
(B) It is pale, completely avascular, dry, and extraordinarily rigid to the touch.
(C) It is entirely black, heavily necrotic, and continuously emits a highly foul odor.
(D) It is characterized throughout by extensive dystrophic calcification and new bone formation.

2007

162. Identify the INCORRECT statement regarding the components of granulation tissue.
(A) It contains a dense, highly intricate network of newly formed, fragile capillaries.
(B) It contains exceptionally abundant populations of active, proliferating fibroblasts.
(C) It features heavily cross-linked type I collagen.
(D) It contains a highly loose, significantly edematized extracellular matrix framework.

2003

163. What physiological requirement causes the rapid induction of angiogenesis during granulation tissue formation?
(A) High metabolic oxygen and nutrient demands of fibroblasts and macrophages
(B) The pathological need to severely and rapidly increase localized hydrostatic pressure to burst the closed wound wide open.
(C) The physiological necessity to immediately and systematically spread localized pathogenic bacteria throughout the host system.
(D) The specific cellular requirement to actively transport large amounts of melanin pigment into the wound for rapid coloration.

2009

164. In a clinical observation of a healing wound, the presence of edematous fluid within the granulation tissue is common. This represents the application of which underlying property of newly formed blood vessels?
(A) They have incredibly tight, fully impermeable and highly specialized intercellular junctions.
(B) They completely lack functional vascular endothelial growth factor (VEGF) receptors entirely.
(C) They are heavily muscled vascular arterioles that actively pump fluid into the local tissue forcefully.
(D) They are structurally immature with leaky interendothelial junctions.

2021

165. How is angiogenesis primarily classified mechanically during tissue repair?
(A) The de novo generation of blood vessels from circulating endothelial stem cells in the void space
(B) The sprouting of new vessels from pre-existing capillaries and venules
(C) The conversion of lymphatic vessels into functional arteries
(D) The formation of vessels directly from differentiating fibroblasts

2018

166. Comparing fibroplasia to angiogenesis in granulation tissue development, which statement is true?
(A) Fibroplasia involves endothelial cell migration, whereas angiogenesis involves collagen deposition.
(B) Fibroplasia is completed within hours, whereas angiogenesis takes years.
(C) Angiogenesis precedes and provides the necessary vascular support for optimal fibroplasia.
(D) Both processes are potently inhibited by Transforming Growth Factor-beta (TGF-β).

2019

167. All of the following are essential steps in angiogenesis, EXCEPT:
(A) Vasodilation in response to nitric oxide and VEGF-induced increased permeability
(B) Proteolytic degradation of the parent vessel basement membrane
(C) Migration and proliferation of endothelial cells toward the angiogenic stimulus
(D) Immediate calcification of the new

Click Here to Buy book on Amazon

endothelial tubes

2002

168. Which molecule is considered the most critical, highly specific inducer of angiogenesis during granulation tissue formation?
(A) Vascular Endothelial Growth Factor (VEGF)
(B) Vasoactive histamine stored in mast cell granules
(C) Circulating acute-phase reactant C-reactive protein
(D) Highly potent chemotactic mediator leukotriene B_4

2016

169. From the abundant presence of myofibroblasts at a wound site roughly 1 to 2 weeks post-injury, one can accurately infer that the wound is undergoing:
(A) Acute hemorrhagic inflammation
(B) Extensive active wound contraction
(C) Initial hemostatic plug formation
(D) Malignant transformation

2005

170. Consider the following statements regarding cellular roles in the proliferation phase of wound repair:
I. Fibroblasts: Synthesis of collagen and extracellular matrix
II. Endothelial cells: Active contraction of the wound edges
III. Myofibroblasts: Expression of α-smooth muscle actin for wound contraction
Which of the above statements are correct?
(A) I and II
(B) II and III
(C) I and III
(D) I

2024

171. What is the precise definition of a myofibroblast?
(A) A fibroblast with contractile smooth muscle cell features
(B) A highly specialized tissue macrophage that has completely engulfed neighboring necrotic muscle fibers.
(C) A terminally differentiated mature skeletal muscle cell that produces exceptionally abundant amounts of collagen.
(D) A specialized vascular endothelial cell that actively forms tight contractile sphincters in newly sprouted capillaries.

2012

172. During the later stages of wound repair, what primarily happens to the dense population of macrophages, fibroblasts, and endothelial cells that characterized the granulation tissue?
(A) They remain permanently active and highly concentrated at the site for life.
(B) They undergo targeted necrosis to provide nutrients for the collagen.
(C) They largely regress via apoptosis, leaving behind a relatively acellular scar.
(D) They migrate into the systemic circulation to settle in the bone marrow.

2003

173. Identify the correct statement concerning the role of macrophages in the proliferation phase.
(A) Macrophages rely solely on active phagocytosis of debris and have no secretory role whatsoever in this phase.
(B) Macrophages directly and continuously synthesize abundant type I collagen to form the main scar framework.
(C) Macrophages actively and completely suppress local angiogenesis in order to prevent subsequent capillary hemorrhage.
(D) They secrete key growth factors like TGF-β and PDGF to recruit fibroblasts.

2008

174. Identify the INCORRECT statement regarding cellular activities during wound contraction.
(A) Wound contraction drastically reduces the surface area of a large defect.
(B) Myofibroblasts utilize an internal network of actin and myosin to exert contractile force.
(C) Wound contraction is driven primarily by the sudden shrinking of the native surrounding bone tissue.
(D) The contractile forces are transmitted to the surrounding tissue via connections to the extracellular matrix.

NODIA

2023

175. What is the direct effect of TGF-β on fibroblasts during the repair process?
(A) It causes immediate, widespread, and massive apoptosis of all tissue fibroblasts in order to completely halt any active fibrosis.
(B) It stimulates fibroblast migration and matrix synthesis while inhibiting degradation.
(C) It induces local tissue fibroblasts to strictly and irreversibly differentiate into functional, oxygen-carrying circulating red blood cells.
(D) It completely prevents tissue fibroblasts

from producing or secreting any functional extracellular matrix structural proteins.

2001

176. In a severely contracted burn wound over a joint, the patient suffers from limited mobility. The application of which specific cell type's excessive activity is clinically evident here?
(A) Eosinophils
(B) Mast cells
(C) Plasma cells
(D) Myofibroblasts

2022

177. How are extracellular matrix components broadly classified based on their structural and functional roles?
(A) Structural proteins, hydrated gels, and adhesive glycoproteins
(B) Intracellular cytoplasmic fluids, inflammatory transudates, and purulent cell-rich exudates
(C) Downstream cellular cyclooxygenase products and active inflammatory lipoxygenase products
(D) Systemic endogenous pyrogens, chemotactic chemokines, and active complement anaphylatoxins

2012

178. Comparing Type I and Type III collagen in the context of wound healing, which statement accurately describes their sequence of appearance?
(A) Type I collagen is initially deposited in high amounts, then completely and systematically replaced by Type III collagen.
(B) Both collagen types are deposited simultaneously in equal amounts and remain in these strict proportions forever.
(C) Type III is deposited first, then replaced by stronger Type I during remodeling.
(D) Type III collagen is exclusively found within cortical bone matrix, while Type I is located exclusively in blood plasma.

2015

179. All of the following are critical functions of the extracellular matrix in tissue repair, EXCEPT:
(A) Directly performing the phagocytosis of invading bacteria
(B) Providing a mechanical support framework for cell anchorage
(C) Acting as an active reservoir for storing latent growth factors
(D) Modulating cell proliferation and differentiation via integrin signaling

2011

180. Consider the following statements regarding extracellular matrix components and their specific characteristics:
I. Elastin: Provides tissues with the ability to recoil after physical deformation
II. Proteoglycans: Highly hydrated gels that confer compressive resistance
III. Fibronectin: A strictly intracellular structural protein that prevents cell movement
IV. Type I Collagen: The primary collagen type found in the delicate, initial 3-day granulation tissue
Which of the above statements are INCORRECT?
(A) I and II
(B) I, II and III
(C) III and IV
(D) II, III and IV

2006

181. From the vital requirement of Vitamin C for the hydroxylation of proline and lysine residues, one can infer that a severe Vitamin C deficiency (scurvy) will directly impair:
(A) The rapid synthesis of preformed vascular histamine
(B) Collagen cross-linking and tensile strength
(C) The complete oxygen-dependent respiratory burst in neutrophils
(D) The systemic cellular production of active arachidonic acid

2011

182. What is the functional advantage of matrix metalloproteinases (MMPs) during the remodeling phase of wound healing?
(A) They continuously and directly stimulate unchecked, rapid fibroblast cellular mitosis at the injury site.
(B) They rapidly and systematically polymerize circulating soluble fibrinogen into a rigid, highly protective blood clot.
(C) They degrade matrix to reshape the scar and clear temporary scaffolding.
(D) They induce permanent, highly pronounced local vasodilation of the surrounding functional microvasculature.

2012

183. What defines adhesive glycoproteins in the extracellular matrix?
(A) Specialized matrix molecules that strictly

Downloaded from www.pdf.tube

and exclusively provide high elasticity and structural recoil to highly dynamic tissues like large blood vessels.
(B) Massive, highly organized physical aggregates of hyaluronic acid and proteoglycans that actively resist high localized tissue compressive forces.
(C) Highly specialized circulating plasma proteins that directly and sequentially initiate the classical or alternative complement cascade pathways.
(D) Proteins (like fibronectin/laminin) connecting ECM elements and cells via integrins

2003

184. Which specific enzyme class is responsible for the crucial cross-linking of collagen fibrils to maximize tensile strength during maturation?
(A) Lysyl oxidases
(B) Matrix metalloproteinases
(C) Cyclooxygenases
(D) Phospholipases

2025

185. Identify the correct statement regarding Transforming Growth Factor-beta (TGF-β) in tissue repair.
(A) It is clinically and experimentally recognized as the most potent endogenous inhibitor of tissue fibrosis known in pathology.
(B) It promotes fibrosis by increasing collagen synthesis and decreasing MMP activity.
(C) It acts primarily and directly on target tissues to induce rapid, widespread enzymatical destruction of the extracellular matrix.
(D) It exclusively and selectively stimulates the rapid proliferation of superficial epithelial cells while systematically killing fibroblasts.

2003

186. Identify the INCORRECT statement regarding Platelet-Derived Growth Factor (PDGF).
(A) It is rapidly and selectively released from the dense alpha granules of platelets immediately upon their physiological activation.
(B) It signals via intracellular cAMP instead of tyrosine kinase receptors.
(C) It actively causes robust, directed migration and rapid mitotic proliferation of surrounding tissue fibroblasts during wound repair.
(D) It serves as a highly potent, broad-spectrum chemoattractant for many diverse inflammatory and stromal cell types in the wound.

2003

187. What is the direct cellular effect when Epidermal Growth Factor (EGF) binds to its receptor (EGFR) on basal keratinocytes?
(A) Receptor tyrosine kinase activation, driving keratinocyte proliferation and migration
(B) Immediate and widespread cellular apoptosis of all regional basal keratinocytes near the injury site.
(C) Sudden, irreversible mechanical rigidification of the lipid cell membrane, completely preventing any cellular migration.
(D) Complete, rapid inhibition of all metabolic and transcriptional activity to systematically conserve vital cellular energy.

NODIA

2004

188. In the treatment of a chronic, non-healing diabetic ulcer, a recombinant form of a specific growth factor is applied to robustly stimulate new blood vessel formation. Which growth factor is conceptually being applied here?
(A) Multi-functional Fibroblast Growth Factor (FGF)
(B) Fibgenic Transforming Growth Factor-beta (TGF-β)
(C) Vascular Endothelial Growth Factor (VEGF)
(D) Mitogenic Epidermal Growth Factor (EGF)

2018

189. How are growth factor receptors broadly classified based on their predominant signal transduction mechanism in tissue repair (e.g., for EGF, VEGF, PDGF)?
(A) G-protein coupled receptors
(B) Intracellular nuclear receptors
(C) Ion channel-linked receptors
(D) Receptor tyrosine kinases

2010

190. Consider the following statements regarding growth factors and their primary cellular targets or roles:
I. VEGF: Stimulates angiogenesis
II. TGF-β: Potent fibrogenic agent
III. EGF: Stimulates epithelial cell proliferation
IV. PDGF: Completely inhibits fibroblast migration
V. FGF: Induces purely cartilage destruction without any other effects
Which of the above statements are correct?
(A) I, II and III
(B) I, II and III
(C) II, III, IV and V
(D) I, III, IV and V

2017

191. All of the following are recognized functions of Fibroblast Growth Factor (FGF), EXCEPT:
(A) Actively stimulating new blood vessel sprouting and angiogenesis
(B) Inhibiting extracellular matrix protein synthesis
(C) Promoting the direct migration of active macrophages and fibroblasts
(D) Stimulating epithelial cell migration to cover open epidermal wounds

2003

192. Which molecule is most appropriately identified as a master regulator that limits the inflammatory response and powerfully drives the transition to fibrosis?
(A) Pro-inflammatory Interleukin-1
(B) Tumor Necrosis Factor-α
(C) Vasoactive peptide bradykinin
(D) Transforming Growth Factor-beta

2011

193. From the widespread distribution of Epidermal Growth Factor Receptors (EGFR) on numerous cell types beyond just the epidermis, what can be inferred about EGF's functional scope?
(A) EGF likely drives proliferation of various epithelial and mesenchymal cells during repair.
(B) EGF physiological activity affects and targets only the most superficial cellular layer of the skin.
(C) EGF biological signaling must be tightly and continuously restricted to prevent systemic tumor growth.
(D) EGF is functionally an obsolete, redundant evolutionary molecule in modern human systems physiology.

2014

194. What is a key clinical advantage of a wound healing by primary intention compared to secondary intention?
(A) It generates an enormously massive, dense fibrous scar that actively protects the underlying tissue like a hard armor.
(B) It completely and flawlessly bypasses the entire cellular and vascular inflammatory stage of wound healing.
(C) It involves a smaller defect, less granulation, and minimal scarring.
(D) It requires absolutely no cellular involvement or contribution from active fibroblasts or secreted structural collagen.

2012

195. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option:
Assertion (A): Wounds healing by secondary intention exhibit significantly more wound contraction than those healing by primary intention.
Reason (R): Secondary intention involves larger tissue defects with greater amounts of granulation tissue and a higher population of contractile myofibroblasts.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2014

196. Which classic example represents a wound that strictly heals by primary intention?
(A) A large, open, infected third-degree burn wound
(B) A massive, deeply burrowing stage IV pressure ulcer on the sacrum
(C) A deeply situated, actively ruptured suppurative abscess cavity
(D) A clean, uninfected surgical incision closed by sutures

2010

197. Identify the correct statement regarding wound healing by secondary intention.
(A) The physical wound edges are tightly and closely opposed by artificial mechanical suturing means.
(B) Abundant granulation tissue fills the large gap from the bottom up.
(C) The overall volume of necrotic cellular debris, inflammatory exudate, and fibrin is exceptionally minimal.
(D) The final connective tissue scar becomes practically and completely invisible within exactly three days.

2004

198. Identify the INCORRECT statement comparing primary and secondary intention healing.
(A) Both primary and secondary intention healing strictly require a well-orchestrated acute inflammatory phase.
(B) Secondary intention healing pathways clinically carry a significantly and dangerously higher risk of secondary infection.

Click Here to Buy book on Flipkart

(C) Primary intention relies solely on epithelial regeneration with no connective tissue repair.
(D) Active wound contraction mediated by specialized myofibroblasts is a classical hallmark feature of secondary intention.

2024
199. What is the direct cause of the intense, robust wound contraction seen in large surface ulcers healing by secondary intention?
(A) The rapid, passive evaporation of water and extracellular fluids directly from the exposed wound surface.
(B) The rapid, abnormal dystrophic crystallization of calcium salts throughout the vascular wound bed.
(C) The progressive shrinkage of dead or dying red blood cells physically trapped within the initial fibrin clot.
(D) Myofibroblasts forming a network that exerts mechanical pull on wound margins

2021
200. Match the wound type in List I with its typical healing intention or characteristic in List II:
List I: (Wound type)
P. Clean surgical incision
Q. Large pressure ulcer
R. Primary intention scar
S. Secondary intention scar
List II: (Healing intention or characteristic)
1. Healing by primary intention
2. Healing by secondary intention
3. Minimal to no wound contraction
4. Significant wound contraction
(A) P-2, Q-1, R-4, S-3
(B) P-1, Q-2, R-3, S-4
(C) P-3, Q-4, R-1, S-2
(D) P-4, Q-3, R-2, S-1

2001
201. How is the process of epithelialization classified in secondary intention compared to primary intention?
(A) It occurs virtually instantly and spontaneously without any active epidermal cellular migration in secondary intention.
(B) It requires extensive epidermal migration and proliferation over a large granulation bed.
(C) It is entirely and permanently absent in secondary intention, relying strictly and solely on dense fibrotic scar tissue.
(D) It strictly and exclusively proceeds from the geometric center of the open wound outwards in secondary intention.

2001
202. Comparing a hypertrophic scar and a keloid, which feature distinguishes a keloid?
(A) It strictly, consistently, and completely regresses spontaneously over a period of a few short months.
(B) It grows beyond the original wound boundaries into healthy tissue.
(C) It is structurally composed primarily of intracellular lipid vacuoles rather than dense extracellular collagen.
(D) It occurs entirely, universally, and directly due to a severe localized or systemic clinical lack of vitamin C.

2002
203. All of the following are recognized local complications that can delay wound healing, EXCEPT:
(A) Adequate, robust vascular supply to the wound area
(B) Presence of foreign bodies
(C) Localized infection in the wound bed
(D) Excessive mechanical motion or tension across the wound

2019
204. Which of the following conditions is most appropriately described as "exuberant granulation tissue" (proud flesh)?
(A) A pathologically compromised wound where active fibroblasts completely fail to produce any structural collagen fibers.
(B) A sudden, massive, and highly dramatic mechanical rupture and separation of a healing abdominal surgical wound.
(C) Excessive granulation tissue protruding above the skin, blocking re-epithelialization
(D) The deep, highly disabling fibrotic contraction of a severe burn scar that permanently limits local joint movement.

2013
205. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option:
Assertion (A): Wound dehiscence is most frequently observed following abdominal surgery rather than limb surgery.
Reason (R): Abdominal wounds are frequently subjected to increased mechanical tension due to coughing, vomiting, or abdominal distension.

Downloaded from www.pdf.tube

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2007

206. What is the clinical advantage of surgical excision in the management of exuberant granulation tissue (proud flesh)?
(A) It permanently, completely, and irreversibly prevents any subsequent cellular or structural wound healing.
(B) It intentionally introduces massive, uncontrolled bacterial colonization to clinically stimulate native immunity.
(C) It removes the physical barrier, letting epidermis migrate and close the defect.
(D) It selectively, biochemically, and completely kills all local tissue myofibroblasts to permanently halt contraction.

2003

207. What defines wound dehiscence?
(A) The pathological overproduction of structurally weak collagen molecules causing a prominently raised scar.
(B) The mechanical separation of previously approximated wound edges
(C) The abnormal, restrictive attachment of internal organs to the abdominal wall via dense fibrous tissue bands.
(D) The complete, highly pathological absence of any localized acute inflammatory response following tissue injury.

2025

208. During a clinical examination of a severe burn survivor, tight, disabling deformities around the elbow joints are noted. What pathological complication of healing is this classically recalled as?
(A) Keloid formation
(B) Exuberant granulation
(C) Wound dehiscence
(D) Wound contracture

2006

209. Identify the correct statement regarding systemic factors that impede wound healing.
(A) Glucocorticoids impede healing by inhibiting inflammation and collagen synthesis.
(B) Uncontrolled hyperglycemia associated with chronic diabetes dramatically and flawlessly accelerates perfect wound regeneration.
(C) Severe, prolonged protein malnutrition specifically, systematically, and dynamically enhances the overall strength of remodeling collagen.
(D) Exceptionally young age drastically, clinically, and universally delays and compromises the physiological rate of tissue repair.

2021

210. Match the pathological aberration of healing in List I with its descriptive feature in List II:

List I: (Pathological aberration)
P. Keloid
Q. Hypertrophic scar
R. Proud flesh
S. Contracture

List II: (Descriptive feature)
1. Raised scar confined to original wound borders
2. Scar tissue growing beyond original wound boundaries
3. Excessive granulation tissue blocking epithelialization
4. Exaggerated wound shrinkage causing structural deformity

(A) P-1, Q-2, R-3, S-4
(B) P-2, Q-1, R-4, S-3
(C) P-2, Q-1, R-3, S-4
(D) P-3, Q-4, R-2, S-1

****

NODIA

ANSWERS

1. (B) To dilute, isolate, and eliminate the inciting agent and prepare the tissue for repair
Acute inflammation serves as a protective vascular and cellular response designed to handle initial tissue injury. By delivering defensive materials directly to the affected site, this physiological process dilutes harmful substances, isolates the damaging agent, eliminates pathogens or dead cells, and establishes the essential biological framework required for subsequent wound healing and tissue regeneration.

2. (A) Damage-associated molecular patterns
Sterile inflammation is initiated when intracellular molecules are released from damaged or dying host cells in the absence of foreign pathogens. These endogenous substances, known as damage-associated molecular patterns, are recognized by pattern recognition receptors on sentinel cells. This interaction triggers an inflammatory cascade without requiring infectious microbes or pathogen-associated molecular patterns.

3. (C) Mechanical trauma initiates inflammation through rapid histamine release from resident tissue mast cells.
Physical injuries like mechanical trauma cause immediate, localized cellular disruption that directly stimulates resident mast cells in the connective tissue. These activated cells rapidly degranulate, releasing preformed vasoactive mediators such as histamine into the surrounding microenvironment. This release initiates downstream vascular changes, including local vasodilation and increased capillary permeability, characterizing early acute inflammation.

4. (D) Autoimmune reactions bypass acute phases to directly initiate granulomatous inflammation.
Autoimmune reactions do not bypass the acute phase of inflammation. Instead, persistent self-antigens continually trigger cycles of acute vascular and cellular responses before transitioning into chronic or granulomatous phases. The sustained immune response eventually leads to chronic tissue remodeling, but the initial response always involves classic acute inflammatory pathways, leukocyte recruitment, and vascular changes.

5. (C) Liquefactive necrosis with cellular dissolution and lipid breakdown
Strong alkaline chemicals cause rapid liquefactive necrosis because they saponify lipids in cell membranes and denature structural proteins. This enzymatic and chemical digestion destroys the cellular framework, leading to complete tissue dissolution and a liquid mass. The extensive damage and release of cellular constituents trigger an intense, immediate inflammatory response in surrounding healthy tissues.

6. (B) Lipopolysaccharide
Lipopolysaccharide is a major structural component of the outer membrane of Gram-negative bacteria. It serves as a classic pathogen-associated molecular pattern that binds with high affinity to Toll-like receptor 4 on host immune cells. This binding activates intracellular signaling pathways, initiating transcription of pro-inflammatory cytokines and driving the acute inflammatory response.

7. (D) Foreign body inflammation
Retained surgical sutures represent non-biodegradable, exogenous materials that cannot be easily cleared by phagocytosis. This persistence triggers foreign body inflammation, a specialized type of chronic inflammation characterized by the accumulation of macrophages and multinucleated foreign body giant cells around the material. This localized response attempts to wall off the insoluble substance from healthy tissues.

8. (A) The elaboration of pathogen-associated molecular patterns
Pathogen-associated molecular patterns are unique biochemical structures found exclusively on microorganisms, such as bacterial lipopolysaccharides or viral nucleic acids. While physical triggers initiate sterile inflammation through damage-associated molecular patterns from dying host cells, only infectious triggers introduce these foreign microbial molecules to stimulate pattern recognition receptors, establishing a unique biological signature for infection.

Click Here to Buy book on Amazon

9. (C) Functio laesa
Loss of function, or functio laesa, is a complex clinical manifestation driven by multiple factors, including mechanical limitations, reflex muscle spasms, and severe pain. Unlike redness, heat, and swelling, which result directly from increased microvascular blood flow and fluid exudation into tissues, loss of function represents a protective behavioral response to avoid further anatomical injury.

10. (A) Both A and R are true and R is the correct explanation of A.
The cardinal sign of heat is caused by increased blood flow carrying core warmth to peripheral tissues. Because internal organs already exist at core temperature, localized vasodilation during deep tissue inflammation cannot raise their temperature above the surrounding baseline. Thus, local heat is a distinct clinical feature of superficial, but not deep visceral, inflammation.

11. (D) Bradykinin
Bradykinin is a highly potent vasoactive peptide generated during the activation of the plasma kinin cascade. It directly binds to specific receptors on local nociceptors, lowering their activation threshold and triggering the sensation of pain. While prostaglandins also sensitize these nerve endings, bradykinin acts as a primary, direct chemical mediator of inflammatory pain.

12. (B) Protein-rich exudate accumulates in the extravascular space.
Inflammatory swelling, or tumor, develops when vascular permeability increases, allowing plasma proteins to escape the microcirculation. This movement alters the osmotic gradient, drawing water into the interstitial tissues. The resulting accumulation of protein-rich fluid, known as an exudate, exceeds the capacity of local lymphatic drainage and physically expands the extracellular space.

13. (C) It encourages host rest of the injured part to prevent further damage.
Loss of function serves as a critical evolutionary adaptation that preserves tissue integrity during healing. By inducing pain and physical limitation upon movement, this symptom forces the host to immobilize and rest the inflamed area. This reduction in physical stress prevents further mechanical disruption, protects delicate repair tissue, and facilitates more efficient cellular regeneration.

14. (D) Arteriolar vasodilation and engorgement of capillary beds
The cardinal sign of redness, or rubor, is caused by the relaxation of vascular smooth muscle in local arterioles. This vasodilation increases blood flow into downstream capillary beds, which become heavily engorged with oxygenated red blood cells. The increased density of erythrocytes within the microcirculation imparts a bright red clinical appearance to the inflamed tissue.

15. (A) Aulus Cornelius Celsus
Aulus Cornelius Celsus was an ancient Roman encyclopedist who documented the early medical knowledge of his era. In his celebrated work, De Medicina, he formally identified and described the four classic clinical signs of acute inflammation. These historical hallmarks—redness, swelling, heat, and pain—remain foundational concepts in modern pathological evaluations of tissue injury.

NODIA

16. (B) Functio laesa was added to the signs by Rudolf Virchow in the 19th century.
While the first four cardinal signs of inflammation were established in antiquity, loss of function was introduced much later. Rudolf Virchow, the pioneer of cellular pathology, added this fifth sign in the nineteenth century to recognize that inflamed tissues suffer compromised biological activity. This completed the classic clinical pentad used to evaluate local inflammatory states.

17. (B) Increased vascular permeability invariably occurs before any arteriolar vasodilation.
In the vascular response of acute inflammation, arteriolar vasodilation occurs first, driven by mediators like histamine. This expansion of the microvascular bed increases blood flow and hydrostatic pressure. Only after this initial vasodilatory phase does vascular permeability increase, permitting fluid to exit the vessels into the interstitial space, which eventually leads to blood stasis.

18. (A) Increased blood viscosity from exudation of protein-rich fluid
Blood stasis during acute inflammation is primarily caused by a dramatic shift in fluid dynamics. As vascular permeability rises, water and plasma proteins escape into the interstitial

tissue. This leaves behind a highly concentrated population of red blood cells, which increases blood viscosity. The thickened blood slows down significantly, allowing leukocytes to marginate along endothelial walls.

19. (C) Poiseuille's law on viscosity and flow rate
Poiseuille's law dictates that fluid flow rate is inversely proportional to fluid viscosity. During acute inflammation, the rapid loss of plasma fluid into the extravascular space concentrates erythrocytes within the microvessels. This hemoconcentration dramatically increases local blood viscosity, causing flow velocity to plunge, which allows leukocytes to move from the central stream toward the endothelium.

20. (D) P-3, Q-4, R-1, S-2
The hemodynamic phases of acute inflammation proceed through distinct mechanisms. Initial transient vasoconstriction relies on neurogenic reflexes. Subsequent arteriolar vasodilation is stimulated by histamine acting on smooth muscle. Increased vascular permeability is driven by endothelial cell contraction in post-capillary venules. Finally, fluid loss causes hemoconcentration and increased blood viscosity, resulting in microvascular stasis.

21. (A) Delayed prolonged response
Mild thermal injury typically induces a delayed prolonged response in vascular permeability. Unlike the immediate transient response seen with chemical mediators like histamine, this reaction begins after a delay of several hours and can persist for days. It involves structural changes and damage to both capillaries and venules, often requiring direct cellular repair.

22. (B) Inflamed beds have higher intravascular hydrostatic pressure and higher interstitial osmotic pressure.
Vasodilation in inflamed tissue increases blood flow and intravascular hydrostatic pressure. Simultaneously, increased vascular permeability allows proteins to escape into the extracellular space, raising the interstitial osmotic pressure. These combined pressure shifts alter normal Starling forces, promoting the continuous movement of fluid out of the microvessels and leading to localized edema.

23. (D) Large muscular arteries, which do not dilate during localized inflammation
Vasodilation during acute inflammation is a localized microvascular phenomenon. While arterioles, capillaries, and post-capillary venules actively dilate and experience structural alterations to facilitate cellular recruitment, large muscular arteries remain unaffected by these local mediators. They maintain their normal physiological caliber, as their primary function is systemic pressure regulation rather than local tissue perfusion management.

24. (A) Transient vasoconstriction, vasodilation, increased permeability, stasis
Immediately following tissue injury, the microcirculation undergoes a highly coordinated sequence of events. A brief, transient neurogenic vasoconstriction of local arterioles occurs first, followed rapidly by arteriolar vasodilation. This increases blood flow and hydrostatic pressure, leading to increased vascular permeability. As fluid exits, hemoconcentration increases blood viscosity, resulting in localized stasis.

25. (C) Intravascular fluid has been lost to the extravascular space.
Localized hemoconcentration in venules is a direct physical consequence of increased vascular permeability. As endothelial junctions widen, protein-rich plasma fluid escapes into the surrounding interstitial tissues. This fluid loss leaves behind a highly concentrated population of red blood cells within the vessel lumen, increasing blood viscosity and signaling that exudation has occurred.

26. (A) It facilitates transport of specific proteins across intact endothelial cells.
Transcytosis provides an active, intracellular pathway for moving large proteins and macromolecular complexes across the vascular barrier. By utilizing specialized vesiculovacuolar organelles, endothelial cells can transport these essential cargo molecules without disrupting their physical intercellular junctions, allowing for highly regulated protein delivery to the inflamed extravascular space while keeping the barrier intact.

27. (D) The reversible widening of intercellular junctions in post-capillary venules
Endothelial cell contraction is a highly regulated, active process where endothelial cells shorten,

Downloaded from www.pdf.tube

creating physical gaps between them. This event is reversible and occurs almost exclusively in post-capillary venules in response to vasoactive mediators. The resulting widening of these intercellular junctions temporarily increases vascular permeability, letting plasma proteins pass into the interstitial space.

28. (B) Histamine
Histamine is the classic vasoactive amine responsible for eliciting the rapid, reversible endothelial cell contraction during the immediate transient response. Upon release from mast cells, it binds to specific receptors on post-capillary venule endothelial cells, triggering cytoskeletal rearrangement. This action widens intercellular junctions, causing an abrupt and short-lived increase in vascular permeability.

29. (C) It results in an immediate sustained response lasting until vessels are repaired.
Direct endothelial injury occurs when severe insults physically destroy endothelial cells, causing immediate cell death and detachment. This structural damage affects all levels of the microcirculation and produces an immediate sustained response. The high vascular permeability persists for hours or days, continuing until the damaged vessels are structurally repaired or replaced by angiogenesis.

30. (C) I and II
Increased vascular permeability in acute inflammation relies on distinct cellular pathways. Endothelial contraction is a rapid, reversible process mediated by vasoactive amines like histamine. In contrast, transcytosis involves the movement of fluid and proteins through intracellular vesicle channels, a pathway stimulated by vascular endothelial growth factor. Endothelial proliferation, however, serves tissue repair rather than permeability.

31. (A) Toxic oxygen species and proteolytic enzymes from leukocytes damage the endothelium.
During the leukocyte adhesion cascade, activated leukocytes marginate and attach to the endothelial surface. In this process, they release toxic oxygen-derived free radicals and proteolytic lysosomal enzymes directly onto the vessel wall. This chemical release causes localized endothelial cell damage and detachment, which increases vascular permeability in the affected microvascular beds.

32. (B) Direct endothelial injury
Severe thermal burns cause immediate, extensive necrosis of endothelial cells, leading to physical disruption of the vascular barrier. This direct endothelial injury allows unrestricted leakage of protein-rich fluid into the extravascular space, resulting in massive blister formation. The leakage represents an immediate sustained response that continues until the structural integrity of the vessels is restored.

33. (B) It is associated with mild thermal injury, radiation, or certain bacterial toxins.
A delayed prolonged response is a pattern of vascular permeability characterized by a late onset and extended duration. This phenomenon is commonly triggered by mild thermal burns, ultraviolet or ionizing radiation, and specific bacterial toxins. The delayed leakage is believed to involve apoptosis or sublethal structural damage of endothelial cells, which takes hours to manifest.

NODIA

34. (A) Greater than 1.020
An inflammatory exudate is characterized by high protein concentration and cellular debris, which significantly increases its density. This high solute load results in a specific gravity that typically exceeds 1.020. This physical property serves as a reliable marker to distinguish it from a transudate, which is a protein-poor ultrafiltrate of plasma.

35. (C) Low specific gravity (< 1.012)
An inflammatory exudate is a fluid that escapes from the microcirculation due to increased vascular permeability. Because the endothelial barrier is compromised, this fluid is rich in plasma proteins, inflammatory cells, and cellular debris. Consequently, an exudate exhibits a high specific gravity, typically exceeding 1.020, rather than the low specific gravity characteristic of transudates.

36. (D) The ratio of pleural fluid protein to serum protein is greater than 0.5.
In clinical practice, Light's criteria are utilized to differentiate pleural effusions. An exudate is characterized by a pleural fluid to serum protein ratio greater than 0.5, reflecting a significant leakage of large proteins through damaged vessel walls. In contrast, transudates maintain a low ratio because they result from hydrostatic or osmotic imbalances.

37. (C) There is hydrostatic or osmotic imbalance with intact endothelial barriers.
The formation of a transudate, such as in non-inflammatory ascites, indicates that the vascular endothelial barrier remains structurally intact. This fluid accumulation is driven strictly by physical forces, specifically elevated hydrostatic pressure or decreased intravascular oncotic pressure. Because there is no active endothelial damage, the resulting fluid remains poor in proteins and inflammatory cells.

38. (B) It dilutes toxins and delivers immunoglobulins and fibrinogen to the damaged area.
The escape of a protein-rich exudate into damaged tissue provides significant defense advantages. The abundant fluid physically dilutes local bacterial and chemical toxins, reducing their cellular toxicity. Simultaneously, it delivers protective immunoglobulins for opsonization and fibrinogen, which polymerizes into a fibrin meshwork that traps microbes and guides subsequent cellular migration and tissue repair.

39. (D) An ultrafiltrate of blood plasma with low protein content from hydrostatic or osmotic imbalance.
A transudate is chemically defined as a non-inflammatory fluid that represents an ultrafiltrate of blood plasma. It occurs due to systemic imbalances in hydrostatic or oncotic pressure across a microvascular wall with normal permeability. Consequently, this fluid is characterized by a low protein concentration, minimal cellular elements, and a low specific gravity.

40. (B) II
A transudate is characterized by a low protein content and minimal cellular debris, resulting in a low specific gravity that is typically less than 1.012. Therefore, associating a transudate with a high specific gravity is incorrect, as high density and elevated solute levels are classic physical hallmarks reserved exclusively for inflammatory exudates.

41. (C) Exudates often contain polymorphonuclear leukocytes during acute phases.
During the acute phase of inflammation, increased vascular permeability allows both proteins and cells to enter the tissue. The resulting inflammatory exudates are highly cellular, typically containing a large concentration of polymorphonuclear leukocytes, specifically neutrophils. These cells actively migrate through the widened endothelial junctions to combat pathogens and clear cellular debris at the injury site.

42. (A) Transudates contain high levels of fibrinogen and clot spontaneously.
Because the vascular endothelial barrier remains intact during transudate formation, large molecular weight plasma proteins like fibrinogen cannot pass into the fluid. Consequently, transudates are protein-poor and completely lack the capacity to form fibrin networks or clot spontaneously. This key characteristic distinguishes them from protein-rich, highly coagulable inflammatory exudates.

43. (D) Leukocyte rolling and transient attachment along the endothelial surface
The upregulation of P-selectin and E-selectin on activated endothelial cells mediates the initial step of the leukocyte adhesion cascade. These molecules bind to carbohydrate ligands on circulating leukocytes with low affinity. This weak, transient tethering forces the rapidly flowing leukocytes to slow down and roll along the endothelial wall, preparing them for firm adhesion.

44. (B) Integrin-mediated firm adhesion
Leukocyte Adhesion Deficiency type 1 is characterized by a genetic deficiency in the common beta-2 subunit of integrins. Because these critical adhesion molecules are absent or non-functional, marginated leukocytes cannot bind stably to their ligands on the endothelial surface. Consequently, the firm adhesion step of the cascade is blocked, preventing leukocyte transmigration into tissues.

45. (C) ICAM-1
Intercellular Adhesion Molecule-1 is a transmembrane glycoprotein belonging to the immunoglobulin superfamily. Expressed on activated endothelial cells, it serves as a specific ligand for beta-2 integrins, such as LFA-1 and Mac-1, present on the surface of leukocytes. This ligand-receptor interaction is essential for mediating the firm and stable adhesion of leukocytes.

Click Here to Buy book on Flipkart

46. (D) L-selectin
Selectins are vital adhesion molecules that regulate the initial rolling of leukocytes. While P-selectin and E-selectin are synthesized and expressed on the surface of activated vascular endothelial cells, L-selectin is constitutively expressed on the surface of circulating leukocytes, including neutrophils and lymphocytes, helping them home to sites of inflammation and lymphoid tissue.

47. (A) Sialyl-Lewis X
The firm adhesion phase of the leukocyte cascade requires high-affinity interactions between leukocyte integrins, such as LFA-1 and Mac-1, and endothelial ligands like VCAM-1. Sialyl-Lewis X, however, is a carbohydrate ligand that binds to selectins. This molecule is essential for the initial rolling phase of the cascade, rather than the subsequent firm adhesion step.

48. (B) Leukocyte transmigration across the endothelial layer and basement membrane
Diapedesis, or transmigration, is the active process where firmly adherent leukocytes squeeze between neighboring endothelial cells to exit the vessel. This migration occurs primarily in post-capillary venules and requires homophilic interactions between adhesion molecules like CD31. Once through the endothelium, the leukocytes secrete collagenases to cross the vascular basement membrane and enter tissues.

49. (B) Chemokines induce a conformational change in integrins to a high-affinity state.
Integrin activation is a rapid, dynamic physiological process rather than a static state. When rolling leukocytes encounter chemokines displayed on the endothelial surface, these signaling molecules bind to G-protein-coupled receptors. This triggers intracellular signals that induce a conformational change in leukocyte surface integrins, converting them into a high-affinity state that enables firm adhesion.

50. (A) I, II and III
The leukocyte adhesion cascade is regulated by specific molecules at each step. P-selectin mediates the initial rolling of leukocytes, while ICAM-1 is crucial for firm adhesion. PECAM-1 coordinates transmigration through endothelial junctions. Sialyl-Lewis X is involved in rolling rather than firm adhesion, and integrins are primary mediators of firm adhesion prior to transmigration.

51. (C) Leukocyte migration along a chemical gradient toward the site of injury
Chemotaxis is a highly directed form of cellular locomotion where leukocytes migrate through tissue toward an injury site. This movement is guided by a concentration gradient of chemoattractants, which can be exogenous bacterial products or endogenous inflammatory mediators. Leukocytes detect these chemical signals via specific surface receptors, directing their cytoskeletal rearrangement toward the source.

52. (D) Leukotriene B_4
Leukotriene B_4 is an endogenous eicosanoid synthesized from arachidonic acid via the lipoxygenase pathway. It is a highly potent chemoattractant that specifically binds to receptors on neutrophils. This activation triggers calcium mobilization and cytoskeletal remodeling, driving the rapid, directed migration of these primary phagocytic cells toward the localized site of tissue injury or infection.

53. (C) Locomotion is driven by actin polymerization at the leading edge of the cell.
Leukocyte locomotion during chemotaxis is an active process dependent on the cytoskeleton. Binding of chemoattractants to G-protein-coupled receptors triggers intracellular calcium signals. This induces rapid actin polymerization at the leading edge of the cell, forming pseudopods. Simultaneously, myosin filaments contract at the trailing edge, physically pulling the leukocyte forward along the chemical gradient.

54. (B) Interleukin-8 (CXCL8) selectively attracts eosinophils during allergic responses.
Interleukin-8 is a classic chemokine belonging to the CXC family. It is a highly potent and selective chemoattractant for neutrophils, rather than eosinophils. Interleukin-8 binds to CXCR1 and CXCR2 receptors on neutrophils, initiating the cytoskeletal changes required for their migration. Eosinophil recruitment, in contrast, is primarily driven by eotaxin and other CC chemokines.

55. (D) Activation of phospholipase C and increased calcium, causing cytoskeletal changes
Binding of chemoattractants to seven-transmembrane G-protein-coupled receptors

NODIA

on leukocytes initiates intracellular signaling cascades. This binding activates phospholipase C, which cleaves membrane lipids to generate second messengers. This pathway induces a rapid release of intracellular calcium. The elevated calcium levels coordinate the actin and myosin interactions necessary for active cellular pseudopod extension and directed locomotion.

56. (A) Neutrophil migration to the infection site will be impaired.
C5a is one of the most powerful endogenous chemoattractants produced by the complement system. If a bacterial pathogen successfully degrades C5a, the chemical gradient required to guide neutrophils is severely disrupted. Consequently, the directed migration of neutrophils toward the site of infection will be impaired, compromising the host's early cellular immune response.

57. (C) Based on the arrangement of conserved cysteine residues
Chemokines are classified into four major subfamilies based on the arrangement of their amino-terminal cysteine residues. In the CXC subfamily, an amino acid separates the first two cysteines, whereas in the CC subfamily, these residues are adjacent. This structural categorization correlates with functional differences, as CXC chemokines primarily target neutrophils, while CC chemokines target monocytes.

58. (A) Exogenous: N-formylmethionyl peptides; Endogenous: Leukotriene B_4
Chemoattractants are categorized by their origin. Exogenous chemoattractants are derived from microbes, such as N-formylmethionyl peptides, which are initiated during bacterial protein synthesis. Endogenous chemoattractants are synthesized by host cells or derived from plasma proteins, with leukotriene B_4 serving as a classic host eicosanoid that recruits inflammatory cells to sites of tissue injury.

59. (D) Extracellular release of lysosomes
Phagocytosis is an intracellular defense process designed to sequester and destroy foreign particles. Its major steps include the opsonization and recognition of the target, pseudopod engulfment to form an intracellular phagosome, and subsequent phagolysosome fusion. The extracellular release of lysosomal enzymes is a pathological event rather than a standard physiological stage of particle engulfment and clearance.

60. (B) Opsonization → Engulfment → Phagolysosome formation → Microbial killing
Phagocytosis proceeds through a highly structured sequence of cellular events. First, the foreign particle is opsonized with host proteins like IgG or C3b to facilitate receptor recognition. The phagocyte then extends pseudopods to engulf the particle, enclosing it within a phagosome. This vacuole fuses with lysosomes, creating a phagolysosome where enzymatic and oxidative microbial killing occurs.

61. (C) It will prevent engulfment of opsonized particles.
The engulfment of opsonized particles is an active physical process that requires rapid rearrangement of the host cell cytoskeleton. This structural movement is driven by actin polymerization, which allows the cell membrane to extend pseudopods around the target. Inhibiting actin with cytochalasin prevents this membrane extension, blocking the engulfment phase of phagocytosis.

62. (D) It coats microbes with molecules recognized by leukocyte receptors to enhance attachment.
Opsonization is a biological adaptation that enhances host defense by coating foreign pathogens with specific proteins, known as opsonins. These molecules, such as IgG antibodies or C3b complement fragments, are recognized by high-affinity receptors on the surface of phagocytes. This binding provides a secure physical attachment, facilitating rapid engulfment and subsequent intracellular destruction.

63. (A) An intracellular vacuole containing an engulfed particle
A phagosome is a specialized cytoplasmic vesicle formed during the engulfment phase of phagocytosis. As a phagocyte extends its cell membrane around a recognized foreign particle, the pseudopods meet and fuse, completely enclosing the target. This membrane-bound vacuole isolates the pathogen from the host cytoplasm, preparing it for subsequent fusion with lysosomes.

NODIA

64. (B) IgG antibodies and the C3b fragment
Immunoglobulin G and the C3b fragment of the complement cascade serve as the primary opsonins in host immunity. These molecules bind directly to the surface of invading microorganisms. Phagocytes express specific surface receptors, such as Fc receptors and CR1, which bind these opsonins. This receptor-ligand interaction triggers the cytoskeletal changes required for engulfment.

65. (B) The cell membrane zippers around the particle to form an enclosed vacuole.
The engulfment phase is an active process requiring energy and cytoskeletal rearrangement. Upon receptor binding, the phagocyte membrane extends pseudopods that wrap around the target. The membrane zippers around the particle through sequential receptor-ligand interactions. The pseudopods then fuse at their tips, forming an enclosed intracellular vacuole known as a phagosome.

66. (A) Fc receptors on phagocytes bind to the Fab portion of IgM antibodies to initiate engulfment.
Fc receptors on phagocytes do not bind to the Fab portion of antibodies. Instead, they are specific for the constant Fc portion of immunoglobulin molecules, particularly IgG, to facilitate opsonization and engulfment. Furthermore, IgM does not bind directly to Fc receptors; its opsonizing activity is mediated indirectly by activating the complement cascade and deposition of C3b.

67. (C) The formation of a phagolysosome, exposing the particle to hydroly...
Once a phagosome is fully internalized, it moves deeper into the cytoplasm, where its membrane fuses with the membranes of lysosomes. This fusion creates a single, combined vacuole called a phagolysosome. This process releases lysosomal acid hydrolases, proteases, and antimicrobial peptides directly into the vacuole, exposing the engulfed pathogen to enzymatic degradation.

68. (D) The generation of superoxide anion via the respiratory burst
Chronic granulomatous disease is caused by inherited mutations in the genes encoding the phagocyte oxidase enzyme complex. This deficiency prevents phagocytes from utilizing molecular oxygen to generate superoxide anions during the respiratory burst. Consequently, the downstream production of reactive oxygen species, such as hydrogen peroxide and hypochlorous acid, is severely compromised, impairing microbial killing.

69. (C) Oxygen-dependent and oxygen-independent mechanisms
Intracellular microbicidal systems within phagocytes are broadly classified based on their requirement for molecular oxygen. Oxygen-dependent mechanisms utilize the respiratory burst to generate highly reactive oxygen species like superoxide and hypochlorous acid. Oxygen-independent mechanisms rely on preformed lysosomal proteins, including lysozyme, lactoferrin, and defensins, which chemically damage the bacterial cell wall or membrane.

70. (A) 4, 1, 2, 3
During the respiratory burst, molecular oxygen is first converted to superoxide anion by phagocyte oxidase. Superoxide then dismutates into hydrogen peroxide. In the presence of myeloperoxidase and a halide, hydrogen peroxide is converted into hypochlorous acid. This sequential chemical pathway generates increasingly toxic reactive oxygen species to destroy engulfed microorganisms within the phagolysosome.

71. (D) Myeloperoxidase
Myeloperoxidase is an essential enzyme that catalyzes the conversion of hydrogen peroxide and chloride ions into hypochlorous acid. Because this pathway requires molecular oxygen and the generation of reactive oxygen intermediates, it is classified as a key component of the oxygen-dependent killing mechanism. Other lysosomal proteins like lysozyme and defensins function independently of oxygen.

72. (A) The H_2O_2-myeloperoxidase-halide system
The hydrogen peroxide-myeloperoxidase-halide system is the most powerful microbicidal mechanism within neutrophils. This oxygen-dependent pathway utilizes myeloperoxidase, an enzyme stored in azurophilic granules, to convert hydrogen peroxide and chloride ions into hypochlorous acid, which is structurally identical to active bleach. This highly reactive oxidant rapidly destroys bacterial cell membranes and proteins.

Downloaded from www.pdf.tube

73. (C) iNOS produces nitric oxide, which reacts with superoxide to form toxic peroxynitrite.
Inducible nitric oxide synthase is upregulated in macrophages upon activation by cytokines or microbial products. The enzyme synthesizes nitric oxide from L-arginine. This free radical reacts rapidly with superoxide anions produced during the respiratory burst to generate peroxynitrite, a highly reactive nitrogen species that inflicts lethal oxidative damage on microbial proteins, lipids, and DNA.

74. (A) They can function in hypoxic environments where the respiratory burst fails.
Oxygen-independent killing mechanisms rely on preformed proteins stored in lysosomal granules rather than the metabolic generation of reactive oxygen species. This independence allows phagocytes to remain highly microbicidal even within ischemic or hypoxic necrotic tissues where oxygen levels are insufficient to support the respiratory burst, ensuring effective host defense under various metabolic conditions.

75. (D) A transient increase in oxygen consumption during phagocytosis to produce ROS
The respiratory burst is a rapid, transient increase in oxygen consumption that occurs when phagocytes are activated. Rather than serving mitochondrial energy production, this cellular oxygen uptake is utilized by the enzyme phagocyte oxidase to generate superoxide anions and other reactive oxygen species. These highly toxic molecules are directed into the phagolysosome to kill pathogens.

76. (B) Neutrophils
Neutrophils are the primary responders during the early hours of acute inflammation. Due to their high abundance in circulation, rapid response to chemokines, and abundant expression of adhesion molecules, they dominate the inflammatory infiltrate within the first six to twenty-four hours. After this period, they undergo apoptosis and are replaced by longer-lived monocytes.

77. (C) They contain bi-lobed nuclei and large, intensely acidophilic granules.
Eosinophils are specialized granulocytes characterized by a distinct multi-lobed, typically bi-lobed nucleus. Their cytoplasm is packed with large, uniform granules that stain intensely with acidic dyes like eosin, appearing bright red or orange under light microscopy. These granules contain basic proteins, such as major basic protein, which are toxic to parasites.

78. (D) They are strictly found in the circulation and never reside in solid tissues.
Mast cells are specialized sentinel cells that do not circulate in the blood. Instead, they reside in vascularized connective tissues, particularly near epithelial barriers and blood vessels. This strategic localization allows them to act as early sensors of tissue damage or pathogen invasion, rapidly releasing histamine and other pro-inflammatory mediators from their cytoplasmic granules.

79. (A) Neutrophils undergo rapid apoptosis in tissue, whereas macrophages survive much longer.
The cellular composition of the inflammatory infiltrate changes over time due to differing lifespans. Neutrophils are short-lived cells that undergo programmed cell death within twenty-four to forty-eight hours of entering tissues. Macrophages, however, are long-lived, survive for weeks, and can replicate locally, allowing them to dominate the site during later phases of inflammation.

80. (B) Both A and R are true but R is not the correct explanation of A.
Eosinophils are recruited during allergic reactions and parasitic infections, and their granules contain major basic protein, which targets helminths. However, the presence of major basic protein does not explain why they are recruited; their selective localization is driven by specific chemokines like eotaxin and cytokines released during IgE-mediated immune responses.

NODIA

81. (B) Granulocytes with basic-staining granules rich in histamine
Basophils are circulating granulocytes characterized by large cytoplasmic granules that bind basic dyes, staining deep blue or purple. These granules are rich in histamine, heparin, and other vasoactive mediators. Basophils share structural and functional similarities with tissue-resident mast cells, participating in allergic responses and immediate hypersensitivity reactions upon activation.

82. (A) Mast cells reside in tissues, while basophils circulate in blood.
Mast cells and basophils share similar functional profiles but reside in different locations. Mast cells are tissue-resident cells located in connective tissue near blood vessels and epithelial surfaces. Basophils, conversely, are circulating granulocytes that make up a small fraction of white blood cells and enter tissues only during active inflammatory responses.

83. (C) Presentation of processed antigens to naive T-cells
Neutrophils are highly specialized for phagocytosis, reactive oxygen species generation, and neutrophil extracellular trap formation to kill pathogens. However, they are not antigen-presenting cells. Antigen presentation to naive T lymphocytes is a complex function performed by professional antigen-presenting cells, such as dendritic cells and macrophages, which link innate and adaptive immunity.

84. (D) A multi-lobulated nucleus with 3 to 5 lobes connected by fine chromatin
A mature, functioning neutrophil features a highly characteristic multi-lobulated nucleus, typically consisting of three to five distinct nuclear lobes connected by thin strands of chromatin. This distinct lobular architecture, which gives rise to their classification as polymorphonuclear leukocytes, increases cellular flexibility, allowing them to migrate easily through tight endothelial junctions into tissues.

85. (C) It is preformed and released immediately upon mast cell stimulation.
Histamine is synthesized and stored within the metachromatic granules of tissue-resident mast cells before any injury occurs. Upon physical or immunological stimulation, mast cells undergo rapid degranulation, releasing preformed histamine into the extracellular space. This immediate availability bypasses the delay of protein synthesis, allowing for an instantaneous vascular response to tissue damage.

86. (B) It allows immediate release of mediators without the delay of transcription.
Storing vasoactive amines like histamine in preformed cytoplasmic granules provides a significant defense advantage. When sentinel cells detect injury or pathogens, these granules undergo rapid exocytosis, releasing their contents. This mechanism bypasses the hours required for gene transcription and protein translation, enabling an immediate microvascular response to dilute and contain local damage.

87. (D) A nitrogenous compound that induces vascular caliber and permeability changes.
Vasoactive amines are biologically active, nitrogen-containing compounds, such as histamine and serotonin, that act on the microcirculation during inflammation. They bind to specific receptors on vascular smooth muscle and endothelial cells, inducing rapid arteriolar vasodilation and increased venular permeability, which facilitates the delivery of fluid and cells to injured tissues.

88. (A) Platelets and enterochromaffin cells
Serotonin, or 5-hydroxytryptamine, is a vasoactive amine stored in dense granules within blood platelets and in mucosal enterochromaffin cells. During vascular injury or platelet aggregation, it is released into the microvessels. In the inflammatory response, serotonin acts to modulate vascular caliber and permeability, contributing to early hemodynamic changes at the site of damage.

89. (A) Binding of antigen to IgE already attached to FcεRI receptors on mast cells
Binding of antigen to IgE already attached to FcεRI receptors on mast cells is correct because antigen-mediated cross-linking of IgE triggers rapid mast cell degranulation and releases histamine, producing vasodilation and increased vascular permeability during acute inflammation. This is the principal mechanism of immediate hypersensitivity reactions. Cyclooxygenase and prostaglandin E_2 act in inflammatory mediator synthesis, while C3b is mainly involved in opsonization rather than mast cell activation.

90. (A) P-4, Q-3, R-1, S-2
Vasoactive amines have distinct cellular origins and vascular effects. Histamine is primarily stored in tissue-resident mast cells and induces arteriolar vasodilation and increased venular permeability. Serotonin is found in blood platelets and can cause localized vasoconstriction or variable caliber changes, depending on the vascular bed and the specific receptor subtypes present in the tissue.

Click Here to Buy book on Amazon

91. (D) Endothelial cell contraction and increased permeability
Binding of histamine to H1 receptors on endothelial cells of post-capillary venules initiates intracellular signaling that leads to cytoskeletal contraction. This active contraction causes the cells to shorten, physically widening the intercellular junctions between them. This structural alteration permits plasma proteins and fluid to escape into the interstitial space, increasing local vascular permeability.

92. (B) The immediate transient response
Inhibiting mast cell degranulation directly blocks the rapid release of preformed vasoactive amines, particularly histamine. This therapeutic intervention specifically dampens the immediate transient response of vascular permeability, which typically peaks within minutes of an allergic stimulus. Bypassing this phase prevents the rapid development of localized tissue edema, redness, and swelling.

93. (C) Cyclooxygenase and lipoxygenase products
Eicosanoids are lipid mediators synthesized from arachidonic acid via two major enzymatic pathways. The cyclooxygenase pathway converts arachidonic acid into prostaglandins, prostacyclin, and thromboxanes, which regulate vascular caliber and pain. The lipoxygenase pathway generates leukotrienes, which mediate leukocyte chemotaxis and bronchoconstriction, and lipoxins, which function as endogenous resolution-promoting factors.

94. (D) Prostaglandins mediate vasodilation/ pain, while leukotrienes mediate chemotaxis/ permeability.
Prostaglandins and leukotrienes are distinct classes of eicosanoids. Prostaglandins, synthesized via the cyclooxygenase pathway, primarily act as vasodilators and sensitize pain receptors. In contrast, leukotrienes, produced by the lipoxygenase pathway, function as powerful chemoattractants for neutrophils and induce endothelial cell contraction in post-capillary venules, thereby increasing vascular permeability.

95. (A) Leukotriene B_4 (LTB_4)
Leukotriene B_4 is synthesized from arachidonic acid via the lipoxygenase pathway, which is initiated by the enzyme 5-lipoxygenase. Other eicosanoids, such as prostaglandins, prostacyclins, and thromboxanes, are generated through the cyclooxygenase pathway. This biochemical distinction explains why cyclooxygenase inhibitors do not suppress, and may even enhance, leukotriene production.

NODIA

96. (B) Prostacyclin (PGI_2)
Prostacyclin, or PGI_2, is a lipid mediator synthesized by vascular endothelial cells via the cyclooxygenase pathway. It acts as a powerful vasodilator and a potent inhibitor of platelet aggregation. These physiological functions directly oppose those of thromboxane A_2, helping to maintain microvascular patency and limit excessive clotting at sites of localized inflammation.

97. (B) Prostaglandin/thromboxane levels will drop, but leukotriene production may continue.
Nonsteroidal anti-inflammatory drugs selectively inhibit the cyclooxygenase enzymes, blocking the conversion of arachidonic acid into prostaglandins and thromboxanes. However, these drugs do not inhibit the 5-lipoxygenase enzyme. Consequently, while prostaglandin levels decrease, the lipoxygenase pathway remains active, allowing the continued synthesis of leukotrienes and potentially shunting arachidonic acid toward that pathway.

98. (A) They act as negative regulators that promote resolution by inhibiting neutrophil recruitment.
Lipoxins are specialized pro-resolving mediators synthesized from arachidonic acid via the lipoxygenase pathway. They serve as endogenous negative regulators of inflammation by inhibiting neutrophil chemotaxis, adhesion, and migration into tissues. This action helps halt the acute inflammatory response, preventing chronic tissue destruction and facilitating the transition to tissue repair.

99. (C) It is a 20-carbon fatty acid released from cell membrane phospholipids by phospholipase A_2.
Arachidonic acid is a twenty-carbon polyunsaturated fatty acid that resides esterified within cell membrane phospholipids. Upon physical or chemical cell stimulation, the enzyme phospholipase A_2 is activated, cleaving and releasing free arachidonic acid into the cytoplasm. Once released, this molecule serves as the essential precursor for the synthesis of eicosanoids.

100. (C) I and III
Thromboxane A_2 promotes platelet aggregation and vasoconstriction, while prostaglandin E_2 is a major mediator of inflammatory pain and fever. Leukotriene B_4, however, does not inhibit neutrophil recruitment; it is a highly potent chemoattractant that actively recruits and activates neutrophils at the site of localized tissue damage.

101. (C) C3a and C5a act as anaphylatoxins, stimulating histamine release.
During complement activation, the cleavage of C3 and C5 proteins releases small fragments, C3a and C5a, known as anaphylatoxins. These peptides bind to receptors on mast cells and basophils, triggering degranulation and the rapid release of histamine. This release increases local vascular permeability and vasodilation, linking complement activation with early acute inflammation.

102. (B) Bradykinin is a potent long-acting vasoconstrictor.
Bradykinin is a highly potent vasoactive peptide, but it does not act as a vasoconstrictor. Instead, it is a powerful, short-acting vasodilator that relaxes vascular smooth muscle. It also contracts non-vascular smooth muscle, increases vascular permeability, and stimulates nociceptors to produce pain, contributing to the hallmark vascular and sensory symptoms of acute inflammation.

103. (D) It triggers coagulation, kinin, and fibrinolytic systems simultaneously.
Hageman factor, or Factor XII, is a key plasma protein activated upon contact with collagen, basement membranes, or foreign surfaces. Once active, it initiates the coagulation cascade, the kinin system, and the fibrinolytic pathway. This central regulatory role links vascular clotting, inflammatory peptide generation, and clot dissolution, coordinating the overall response to tissue injury.

104. (A) The fibrinolytic system
The fibrinolytic system acts as a key regulatory pathway that limits excessive intravascular coagulation. The enzyme plasmin cleaves fibrin clots, generating soluble fibrin degradation products. These degradation products act to increase microvascular permeability and modulate leukocyte recruitment, helping to manage localized edema and facilitate tissue repair during the resolution phase of inflammation.

105. (C) They circulate as inactive precursors requiring proteolytic cleavage.
Plasma protein-derived mediators, including the complement, kinin, coagulation, and fibrinolytic cascades, circulate in the bloodstream as inactive zymogens. This physiological state prevents inappropriate, systemic inflammation. Upon tissue injury, localized proteolytic cleavage activates these factors, triggering cascade reactions that generate highly active inflammatory mediators restricted primarily to the site of damage.

106. (B) Bradykinin induces pain, whereas histamine generally does not.
Bradykinin and histamine both increase vascular permeability and cause vasodilation, but bradykinin is unique in its ability to directly stimulate nociceptors and induce pain. Histamine is a preformed vasoactive amine stored in mast cells, whereas bradykinin is a peptide cleaved from kininogens by kallikrein. Bradykinin acts as a key mediator of pain during inflammation.

107. (A) Synthesizing prostaglandins from arachidonic acid
Plasmin is a multifunctional fibrinolytic enzyme that cleaves fibrin clots into degradation products, activates Hageman factor, and splits complement protein C3 to generate active anaphylatoxins. However, the synthesis of prostaglandins from arachidonic acid is mediated by cyclooxygenase enzymes rather than plasmin, which has no direct role in the enzymatic pathways of lipid mediator production.

108. (C) C3
The complement system consists of soluble proteins activated by three distinct cascades. The classical, alternative, and lectin pathways utilize different initial triggers but converge at the critical step of C3 activation. Cleavage of C3 by C3 convertase produces active fragments that drive opsonization, leukocyte recruitment, and the assembly of the membrane attack complex to eliminate pathogens.

Downloaded from www.pdf.tube

109. (D) Recruitment of neutrophils via chemotaxis and increased permeability.
Complement fragment C5a serves as a highly potent anaphylatoxin and chemoattractant during acute inflammation. Once generated, it acts directly on vascular endothelial cells to increase permeability and promotes the recruitment and activation of neutrophils. This fragment stimulates leukocyte chemotaxis, adhesion, and the release of inflammatory mediators, rather than suppressing leukocyte functions or directly forming blood clots.

110. (B) II and IV
Interleukin-1 is a key pro-inflammatory cytokine that stimulates the hypothalamus to induce fever, rather than inhibiting it. Interleukin-10 is an anti-inflammatory cytokine that suppresses immune responses, rather than stimulating acute inflammation. In contrast, tumor necrosis factor promotes selectin expression, and interleukin-6 drives hepatic acute-phase protein synthesis, making the other statements biochemically correct.

111. (B) A small secreted protein regulating host immune and inflammatory responses.
Cytokines are small, secreted proteins produced by many cell types, primarily activated lymphocytes, macrophages, and dendritic cells. They function as critical molecular messengers that regulate the intensity, duration, and direction of host immune and inflammatory responses. They are distinct from lipid mediators, large clotting proteins, or intracellular organelles involved in microbial digestion.

112. (D) Tumor Necrosis Factor (TNF) and Interleukin-1 (IL-1)
Tumor necrosis factor and interleukin-1 are the primary pro-inflammatory cytokines that coordinate the systemic acute-phase response. Released mainly by activated macrophages, they travel through the bloodstream to act on the hypothalamus to induce fever, stimulate the bone marrow to release leukocytes, and prompt the liver to synthesize acute-phase proteins like C-reactive protein.

113. (A) TNF induces endothelial activation, promoting leukocyte adhesion and coagulation.
Tumor necrosis factor exerts potent local effects by inducing endothelial activation. This process upregulates the expression of endothelial adhesion molecules, such as selectins and integrin ligands, which facilitates leukocyte recruitment and adhesion. Additionally, it increases vascular permeability and promotes local coagulation, rather than suppressing adhesion molecule expression or causing widespread local macrophage apoptosis.

114. (C) TNF limits bone marrow release, causing severe chronic leukopenia as a primary acute phase response.
Systemic cytokine release during acute inflammation causes fever via hypothalamic action, stimulates liver synthesis of acute-phase proteins, and promotes the release of leukocytes from the bone marrow, which typically results in leukocytosis. Tumor necrosis factor does not limit bone marrow release to cause chronic leukopenia as a primary acute-phase response; instead, it stimulates leukocyte production.

NODIA

115. (A) Fibrinogen coating red blood cells, causing rouleaux formation
During the acute-phase response, systemic inflammatory cytokines stimulate hepatocytes to synthesize and secrete fibrinogen into the bloodstream. Elevated levels of circulating fibrinogen coat the negative charges on erythrocyte membranes, which reduces their mutual repulsion and promotes the formation of stacks called rouleaux. These heavier cellular aggregates sediment more rapidly, leading to an elevated erythrocyte sedimentation rate.

116. (D) It will decrease endothelial activation, reducing leukocyte infiltration and joint damage.
In rheumatoid arthritis, tumor necrosis factor drives joint destruction by activating endothelial cells to recruit inflammatory cells. Administering a neutralizing biologic drug counteracts this process, decreasing endothelial adhesion molecule expression and chemokines. This diminishes leukocyte infiltration into the joint space and slows progressive joint damage, rather than increasing leukocyte recruitment or causing rapid tissue ankylosis.

117. (B) Chemokines direct leukocyte migration, whereas TNF and IL-1 activate endothelium and systemic responses.
Chemokines are a specialized subfamily of small, structurally related proteins that primarily function to direct leukocyte migration and homing via chemotactic gradients. In contrast,

major pro-inflammatory cytokines like tumor necrosis factor and interleukin-1 have broader roles, primarily acting to activate vascular endothelial cells, induce systemic acute-phase responses, and stimulate the production of other inflammatory mediators.

118. (C) It involves high vascular permeability, letting fibrinogen escape and polymerize into fibrin.
Fibrinous inflammation is characterized by a significant increase in vascular permeability, which allows large molecules like fibrinogen to escape the blood vessels. Once in the extracellular space, fibrinogen is converted into insoluble fibrin and forms a dense meshwork. This distinguishes it from serous inflammation, which involves a protein-poor fluid transudate with minimal fibrin deposition.

119. (D) It primarily consists of a cell-free, low-protein ultrafiltrate.
Purulent or suppurative inflammation is characterized by the production of pus, which is a thick, protein-rich exudate containing abundant neutrophils, necrotic cells, and edema fluid. It is typically caused by pyogenic bacteria like Staphylococcus species. It is not a cell-free, low-protein ultrafiltrate; that description applies specifically to serous transudates rather than purulent exudates.

120. (B) I, II, III and IV
Serous inflammation features watery, protein-poor fluid. Fibrinous inflammation involves extensive fibrin meshwork deposition. Suppurative inflammation produces an exudate rich in neutrophils and debris. Catarrhal inflammation involves mucus-producing epithelial surfaces. Hemorrhagic inflammation, however, is characterized by significant vascular damage allowing red blood cells to enter tissues, making the description of complete absence of red blood cells incorrect.

121. (B) The neutrophil
An abscess is a localized collection of purulent inflammatory tissue. The thick, opaque, yellow-green fluid, known as pus, consists of dead and dying neutrophils, necrotic parenchymal cells, and liquefied tissue debris. Neutrophils are the predominant inflammatory cells recruited in response to pyogenic infections, driving the enzymatic liquefaction of tissues and the formation of purulent exudate.

122. (B) It undergoes organization, causing fibroblast ingrowth and fibrous adhesions.
When fibrinous exudate in a body cavity like the pericardium or pleura is not cleared by macrophages, it undergoes organization. During this process, fibroblasts and capillaries grow into the fibrin meshwork, leading to collagen deposition and the formation of fibrous adhesions. This can structurally restrict organ movement and permanently compromise the normal physiological function.

123. (A) Superficial inflammation of mucous membranes with excessive mucus secretion
Catarrhal inflammation is a morphological pattern characterized by superficial inflammation of mucous membranes, which leads to excessive secretion of mucus. This response typically occurs in epithelial linings rich in mucin-producing goblet cells, such as the upper respiratory tract. It does not involve severe parenchymal tissue necrosis, hemorrhage, or the extensive formation of ulcerated caseous lesions.

124. (C) Serous inflammation
The fluid inside a simple skin blister caused by a mild burn is a classic example of serous inflammation. This pattern is characterized by the accumulation of a thin, watery, protein-poor fluid transudate. This fluid is derived from blood plasma or secretions of local mesothelial cells and lacks abundant neutrophils, fibrin meshwork, or erythrocytes.

125. (D) It implies vascular injury with erythrocyte leakage into tissue.
Hemorrhagic inflammation represents a severe morphological pattern indicating extensive vascular injury. This damage allows erythrocytes to leak directly into the inflamed parenchymal tissue and extracellular spaces, giving the exudate a bloody appearance. It is not a mild response, nor is it devoid of active leukocytes or caused by a simple dietary iron deficiency.

126. (C) Suppurative inflammation inevitably heals by first intention without any scarring.
Suppurative or purulent inflammation involves extensive tissue destruction, cellular necrosis, and pus formation. Because of this substantial tissue loss, suppurative lesions cannot heal by first intention and instead require healing by secondary intention, which leads to granulation tissue formation and scarring. The other

Click Here to Buy book on Flipkart

statements regarding ulcers, serous exudate origins, and fibrinous inflammation sites are correct.

127. (B) Persistent inciting stimuli or recurrent acute inflammatory episodes
Chronic inflammation arises when the acute inflammatory response cannot be resolved. This shift is driven primarily by persistent inciting stimuli, such as difficult-to-eradicate microbes, prolonged exposure to toxic agents, or self-antigens causing autoimmune reactions. It is also stimulated by recurrent acute inflammatory episodes, rather than rapid phagocytosis, absence of blood supply, or histamine release.

128. (D) They are secreting specific antibodies against persistent antigens.
Plasma cells in chronic inflammatory tissue are terminally differentiated B lymphocytes. Their primary physiological function in this environment is to continuously synthesize and secrete specific antibodies directed against persistent antigens. These cells do not engage in phagocytosis, do not synthesize collagen for scar formation, and do not lay down the initial fibrin meshwork within the wound.

129. (A) Chronic features mononuclear cells; acute features primarily polymorphonuclear cells.
Indeed, acute and chronic inflammation differ significantly in their predominant cellular infiltrates. Acute inflammation is characterized primarily by the recruitment of polymorphonuclear leukocytes, specifically neutrophils, which act as the first line of defense. In contrast, chronic inflammation is dominated by mononuclear cells, including macrophages, lymphocytes, and plasma cells, which coordinate persistent adaptive immune responses.

130. (A) Bone marrow stem cell → Blood monocyte → Tissue macrophage → Activation
The macrophage life cycle begins with hematopoietic stem cells in the bone marrow, which differentiate into monocytes and enter the bloodstream. These monocytes migrate from the circulation into tissues, where they mature into resident or inflammatory macrophages. Upon encountering specific stimuli, such as cytokines or bacterial products, they undergo activation to perform specialized effector functions.

131. (A) Intense, diffuse neutrophilic infiltration and abundant pus formation
Chronic inflammation is defined histologically by mononuclear cell infiltration, progressive tissue destruction mediated by inflammatory cells, and attempts at healing through connective tissue replacement, which involves angiogenesis and fibrosis. Intense neutrophilic infiltration and abundant pus formation are instead characteristic hallmark features of acute suppurative inflammation, rather than the long-term chronic inflammatory process.

132. (D) Macrophages secrete IL-12 activating T-cells; T-cells secrete IFN-gamma activating macrophages.
Indeed, bidirectional cross-talk between macrophages and lymphocytes drives and sustains chronic inflammation. Activated macrophages present antigens and secrete interleukin-12, which stimulates T lymphocytes to proliferate and differentiate. In turn, activated T-cells secrete interferon-gamma, a potent cytokine that activates macrophages and enhances their phagocytic and secretory capacities, creating a self-perpetuating loop of chronic tissue inflammation.

133. (B) The tissue suffers collateral damage from released reactive oxygen species, proteases, and cytokines.
Indeed, persistent activation of macrophages in chronic inflammation leads to continuous release of biologically active products. These substances include reactive oxygen species, proteases, and pro-inflammatory cytokines, which cause significant collateral damage to the surrounding healthy parenchymal tissues. This ongoing destruction prevents perfect structural regeneration, rather than promoting healing or resulting in sterile tissue isolation.

134. (C) They provide continuous phagocytosis, antigen presentation, and repair signaling.
Indeed, the accumulation of macrophages during prolonged infections provides essential host defense and repair functions. These cells continuously clear pathogens and cellular debris through phagocytosis, present antigens to lymphocytes to sustain adaptive immunity, and secrete growth factors that initiate tissue repair and angiogenesis. They do not bypass adaptive immunity or rely on preformed histamine releases.

Downloaded from www.pdf.tube

135. (D) A focal accumulation of epithelioid macrophages, often surrounded by lymphocytes
A granuloma is a distinct morphological pattern of chronic inflammation characterized by a focal accumulation of activated macrophages that have developed an epithelioid appearance. These epithelioid macrophages are typically surrounded by a collar of mononuclear leukocytes, mainly lymphocytes, and occasionally plasma cells. Granulomas form to contain persistent, difficult-to-eliminate foreign or infectious offending agents.

136. (A) Mycobacterium tuberculosis
Indeed, Mycobacterium tuberculosis is the prototypic infectious pathogen that causes caseating granulomatous inflammation. The immune response to this intracellular bacterium leads to granuloma formation, where the central core undergoes caseous necrosis, presenting as a structured acellular, cheesy debris. This distinct necrotic feature differentiates it from non-caseating granulomas or typical acute suppurative bacterial infections.

137. (B) Activated macrophages with abundant cytoplasm resembling epithelial cells
Epithelioid cells within a granuloma are activated macrophages that have undergone structural transformation. They acquire abundant, pale pink cytoplasm and elongated nuclei, making them resemble epithelial cells under microscopic examination. These cells are specialized for secretory and containment functions rather than active phagocytosis, and they are not true epidermal or migrated mucosal epithelial cells.

138. (B) Multinucleated giant cells divide rapidly via mitosis to populate the granuloma.
Multinucleated giant cells in granulomas are formed by the physical fusion of multiple activated macrophages, rather than by mitotic division. Once fused, these cells do not undergo mitosis to populate the granuloma. Langhans giant cells characteristically show peripheral nuclei in a horseshoe pattern, while foreign body giant cells exhibit haphazardly scattered nuclei within their cytoplasm.

139. (A) T-cell cytokine stimulation, particularly Interferon-gamma
The development of immune granulomas depends on a cell-mediated T-cell immune response. T lymphocytes activated by persistent antigens secrete cytokines, particularly interferon-gamma. This cytokine acts as the primary signal that stimulates macrophages to aggregate, undergo transformation into epithelioid cells, and fuse to form multinucleated giant cells, rather than circulating C3a anaphylatoxins.

NODIA

140. (A) 2, 3, 1, 4
A mature tuberculous granuloma exhibits a highly organized concentric architecture. The innermost core consists of caseous necrosis, which is surrounded by a layer of activated epithelioid macrophages and multinucleated Langhans giant cells. This is enclosed by a collar of lymphocytes, and the outermost rim is composed of fibroblastic proliferation and fibrous collagen deposition.

141. (D) Immune and foreign body granulomas
Indeed, granulomas are broadly classified pathologically into immune granulomas and foreign body granulomas based on their underlying pathogenesis. Immune granulomas are driven by T-cell-mediated immune responses to persistent, insoluble antigens. Foreign body granulomas form in response to inert particles, such as talc or sutures, in the absence of a specific cell-mediated immune response.

142. (C) Peripheral ring of nuclei with foamy, lipid-rich cytoplasm
Touton giant cells are a distinct type of multinucleated giant cell characteristically associated with lipid disorders, such as xanthomas. They are defined by a peripheral ring of nuclei surrounding a central area of cytoplasm, with a highly characteristic foamy, lipid-rich outer cytoplasm. This feature differentiates them from other giant cells like Langhans or foreign body types.

143. (B) Acute appendicitis
Granulomatous inflammation is a specialized form of chronic inflammation seen in specific conditions, including leprosy, syphilis, and sarcoidosis. In contrast, acute appendicitis is characterized by acute inflammation, dominated by vascular congestion, edema, and a massive influx of neutrophils into the muscularis propria, rather than the formation of organized mononuclear epithelioid granulomas.

144. (D) Labile cells
Labile cells are classified as cells that continuously cycle and multiply to replace dead or dying cells

that are constantly being destroyed or shed. These populations possess a high regenerative capacity and are found in tissues with high physiological turnover, such as the squamous epithelium of the skin, oral cavity, and mucosal linings of the gastrointestinal tract.

145. (A) Being mitotic-incapable, destroyed cells are replaced by fibrous scar tissue.
Neurons and cardiac myocytes are permanent cells that have exited the cell cycle and are completely incapable of mitotic division in postnatal life. Following a severe injury, destroyed cells in these tissues cannot regenerate. Instead, the area is healed through scar formation, where fibroblasts proliferate and deposit dense collagen, leading to permanent loss of functional tissue.

146. (C) They rest in G0 but enter the cell cycle to regenerate tissue after injury.
Stable or quiescent cells, such as hepatocytes, maintain a very low rate of replication under normal physiological conditions and reside in the G0 stage of the cell cycle. However, they retain the capacity to re-enter the cell cycle and proliferate rapidly in response to injury or tissue loss, facilitating organized parenchymal regeneration and restoring organ architecture.

147. (A) Cells with low normal replication that divide in response to stimuli
Stable cells are defined as cellular populations that exhibit low baseline replicative activity under normal conditions but can be stimulated to undergo rapid mitosis following tissue damage. These cells reside in the G0 phase of the cell cycle but remain fully capable of re-entering the cycle to regenerate the damaged parenchyma, as exemplified by glandular organs like the liver.

148. (D) Epidermal squamous skin cells
Epidermal squamous skin cells represent a classical example of a labile cell population. These cells undergo continuous, rapid physiological replication throughout life to replace superficial cells that are constantly being shed or destroyed. In contrast, renal tubular cells are stable cells, while skeletal myocytes and central nervous system neurons are permanent cells with no regenerative mitotic capacity.

149. (B) Stromal framework destruction leads to scarring rather than regeneration.
Even in tissues composed of labile or stable cells, perfect structural regeneration requires an intact extracellular matrix. If the stromal framework is severely damaged or completely destroyed, the proliferating cells lack the scaffolding necessary for organized architectural restoration. This leads to haphazard cell deposition and healing via fibrotic scarring rather than organized parenchyma regeneration.

150. (A) Both A and R are true and R is the correct explanation of A.
Hepatocytes are stable cells with a high capacity to regenerate. However, severe liver injury that destroys the reticulin stromal framework prevents organized hepatocyte replication. Lacking this scaffolding, the replicating cells form disorganized nodules separated by dense fibrous collagen scars. This structural disruption is the hallmark of cirrhosis, making both the assertion and reason true and linked.

151. (D) They are terminally differentiated and lack the capacity to enter the mitotic cell cycle.
Adult cardiac myocytes are classified as permanent cells because they are terminally differentiated and have permanently left the cell cycle. Because they lack the capacity to enter mitosis, dead cardiac tissue following myocardial infarction cannot be replaced by new myocytes. Instead, the injured area must heal via fibrosis, leading to a permanent collagenous scar.

152. (A) Proliferation
Early surgical suturing approximates wound edges and minimizes the physical gap. This directly facilitates the proliferative phase of tissue repair by reducing the amount of granulation tissue and cell division needed to bridge the defect. Minimizing this gap accelerates re-epithelialization and collagen deposition, promoting efficient and rapid wound closure with minimal overall scar tissue.

153. (B) Hemostasis → Inflammation → Proliferation → Remodeling
Wound healing is a highly organized, dynamic process divided into four chronological phases. Immediately after injury, hemostasis occurs to control bleeding. This is followed by inflammation to clear pathogens, proliferation to form new granulation tissue and re-epithelialize the defect,

Click Here to Buy book on Amazon

and finally, remodeling to cross-link collagen and maximize the final tensile strength of the scar.

154. (B) Angiogenesis and fibroblast collagen synthesis
The inflammation phase focuses on clearing debris and pathogens via neutrophils and macrophages. In contrast, the subsequent proliferation phase is heavily dominated by constructive cellular activities. These activities include angiogenesis, which sprouts new blood vessels to supply the healing tissue, and fibroblast proliferation and migration, which drives collagen synthesis to form a cellular granulation tissue.

155. (A) Extensive degradation of excess extracellular matrix
Hemostasis is the immediate phase of tissue repair, aimed at halting bleeding. It involves local vasoconstriction, platelet aggregation to form a temporary plug, and activation of the coagulation cascade to produce a stable fibrin clot. Extensive degradation of excess extracellular matrix is not a goal of hemostasis, occurring instead during the much later remodeling phase.

156. (C) Replacing type III collagen with stronger type I and cross-linking
The remodeling or maturation phase of wound healing is a prolonged stage that can last for months or years. During this phase, the temporary and delicate type III collagen deposited in early granulation tissue is progressively replaced by stronger type I collagen. These fibers are structurally cross-linked and rearranged to maximize the final tensile strength of the scar.

157. (D) Remodeling improves tensile strength but never reaches pre-injury perfection.
Indeed, although the remodeling phase significantly improves the tensile strength of a healing wound through collagen cross-linking and reorganization, a fully mature scar never achieves the strength of uninjured native skin. It generally plateaus at approximately 70-80% of original strength after several months, reflecting the inherent structural limitations of repaired dermal connective tissue.

158. (C) They clear necrotic debris and secrete growth factors initiating the proliferative phase.
Macrophages play a critical dual role during the inflammatory and early proliferative stages of tissue repair. They arrive early at the injury site to phagocytose necrotic debris, pathogens, and aged neutrophils. Furthermore, they secrete key growth factors, such as TGF-beta and PDGF, which recruit fibroblasts and stimulate the angiogenesis required to initiate the proliferative phase.

159. (B) Period characterized by granulation, epithelialization, and contraction
Indeed, the proliferative phase is a crucial stage of wound healing characterized by the formation of highly vascularized granulation tissue, re-epithelialization of the epidermal surface, and wound contraction. During this period, active fibroblasts deposit collagen while newly sprouted blood vessels supply nutrients, reducing the size of the wound defect and restoring anatomical tissue continuity.

160. (A) P-2, Q-3, R-1, S-4
Wound healing stages map directly to their hallmark biological events. Hemostasis is characterized by rapid fibrin clot formation to control bleeding. Inflammation involves the infiltration of neutrophils and macrophages to clear the wound. Proliferation features granulation tissue formation with fibroblasts and angiogenesis, while remodeling involves collagen cross-linking and contraction to mature the final scar.

NODIA

161. (A) Pink-to-red, soft, granular, bleeding easily upon minor trauma.
Healthy granulation tissue is characterized macroscopically by its vibrant pink-to-red, soft, and granular appearance. This distinct color and texture result from the abundant, newly formed capillaries and loose, edematous extracellular matrix. Because these newly formed microvessels are delicate and thin-walled, the tissue bleeds easily upon minor physical trauma or gentle touch.

162. (C) It features heavily cross-linked type I collagen.
Granulation tissue is a temporary scaffolding characterized by proliferating fibroblasts, an abundant network of fragile new capillaries, and a loose, edematous extracellular matrix. At this early stage, it features delicate, loosely organized type III collagen rather than heavily cross-linked type I collagen, which is deposited later during the remodeling phase to mature the scar.

163. (A) High metabolic oxygen and nutrient demands of fibroblasts and macrophages
Indeed, the rapid induction of angiogenesis during granulation tissue formation is driven by the high metabolic oxygen and nutrient demands of active cells at the wound site, particularly migrating fibroblasts and macrophages. The newly sprouted capillaries supply these essential metabolic substrates and clear waste products, ensuring optimal cell survival, proliferation, and structural collagen deposition.

164. (D) They are structurally immature with leaky interendothelial junctions.
The prominent edematous fluid commonly observed within healthy granulation tissue occurs because the newly sprouted blood vessels are structurally immature. These nascent microvessels feature leaky interendothelial junctions and have not yet developed a complete basement membrane or peri-endothelial cell support. This high permeability allows proteins and fluids to escape easily into the extracellular space.

165. (B) The sprouting of new vessels from pre-existing capillaries and venules
Indeed, angiogenesis during tissue repair is primarily classified as the sprouting of new blood vessels from pre-existing capillaries and postcapillary venules. This multi-step process involves the localized degradation of the parental basement membrane, migration of endothelial cells toward a growth factor gradient, their proliferation, and subsequent maturation into functional, lumen-bearing microvascular tubes.

166. (C) Angiogenesis precedes and provides the necessary vascular support for optimal fibroplasia.
Angiogenesis and fibroplasia are closely coupled processes during granulation tissue development. Angiogenesis must precede and accompany fibroplasia because the newly sprouted capillaries are essential to provide the oxygen, glucose, and amino acids required by active fibroblasts. Without this robust vascular support, fibroblasts cannot survive, migrate, or synthesize the collagen matrix necessary for tissue repair.

167. (D) Immediate calcification of the new endothelial tubes
Angiogenesis is a complex process involving nitric oxide-mediated vasodilation, increased permeability, proteolytic degradation of the parental basement membrane, and the migration and proliferation of endothelial cells. Immediate calcification of the new endothelial tubes is not a step in angiogenesis; calcification is a pathological process that does not occur during physiological blood vessel sprouting in wounds.

168. (A) Vascular Endothelial Growth Factor (VEGF)
Vascular Endothelial Growth Factor is the most critical and highly specific inducer of angiogenesis during granulation tissue formation. Released in response to local tissue hypoxia, it binds to tyrosine kinase receptors on endothelial cells, stimulating their migration, proliferation, and survival. It is distinct from preformed histamine, acute-phase reactants, or chemotactic lipid mediators like leukotriene B_4.

169. (B) Extensive active wound contraction
The abundance of myofibroblasts at a wound site roughly 1 to 2 weeks post-injury indicates that the wound is undergoing active wound contraction. Myofibroblasts express α-smooth muscle actin, allowing them to exert strong contractile forces that pull the wound margins together, significantly reducing the overall surface area of the original tissue defect.

170. (C) I and III
During the proliferation phase of wound healing, fibroblasts are responsible for synthesizing collagen and the extracellular matrix scaffolding. Myofibroblasts express α-smooth muscle actin, which drives the active contraction of the wound edges. Endothelial cells participate in angiogenesis to form new capillaries rather than contracting the wound, making the second statement biochemically incorrect.

171. (A) A fibroblast with contractile smooth muscle cell features
A myofibroblast is a specialized, activated fibroblast that has acquired structural and functional features of contractile smooth muscle cells. These cells express α-smooth muscle actin in their cytoskeleton, enabling them to exert mechanical force. They play a vital role in wound closure by contracting the margins of large tissue defects, particularly in secondary intention healing.

Downloaded from www.pdf.tube

172. (C) They largely regress via apoptosis, leaving behind a relatively acellular scar.
As granulation tissue matures into a stable scar during the later stages of wound repair, the highly cellular environment undergoes a dramatic shift. The dense populations of active macrophages, fibroblasts, and endothelial cells largely regress via targeted apoptosis. This process transforms the vascular granulation tissue into a pale, relatively acellular, and collagen-rich fibrous scar.

173. (D) They secrete key growth factors like TGF-β and PDGF to recruit fibroblasts.
During the proliferation phase, macrophages function as critical regulators of tissue repair beyond their phagocytic role. They synthesize and secrete key growth factors, particularly transforming growth factor-beta and platelet-derived growth factor. These molecular signals recruit fibroblasts to the wound site, stimulate their proliferation, and drive the deposition of extracellular matrix, while also promoting local angiogenesis.

174. (C) Wound contraction is driven primarily by the sudden shrinking of the native surrounding bone tissue.
Wound contraction is an active physiological process that significantly reduces the size of large tissue defects. It is driven by contractile myofibroblasts containing an internal cytoskeleton of actin and myosin, which transmits pulling forces to the surrounding extracellular matrix. It is not caused by the shrinking of surrounding bone tissue, making that assertion incorrect.

175. (B) It stimulates fibroblast migration and matrix synthesis while inhibiting degradation.
Indeed, transforming growth factor-beta acts as a potent fibrogenic agent during tissue repair. It directly stimulates fibroblast migration to the wound site and upregulates the transcription of genes encoding collagen and other extracellular matrix components. Simultaneously, it decreases matrix degradation by downregulating metalloproteinases in the area and upregulating tissue inhibitors of metalloproteinases, promoting net fibrosis.

176. (D) Myofibroblasts
In a severely contracted burn wound resulting in limited joint mobility, the excessive activity of myofibroblasts is clinically evident. These specialized cells express α-smooth muscle actin, allowing them to pull the edges of the wound together. Overactivity or prolonged presence of myofibroblasts in large wounds leads to pathological contractures, causing severe, disabling joint deformities.

177. (A) Structural proteins, hydrated gels, and adhesive glycoproteins
The extracellular matrix is a complex macromolecular network broadly classified into three structural and functional categories. These are structural fibrous proteins like collagen and elastin that provide tensile strength and recoil, water-hydrated gels like proteoglycans and hyaluronan that resist compression, and adhesive glycoproteins like fibronectin and laminin that connect the tissue cells to the matrix.

178. (C) Type III is deposited first, then replaced by stronger Type I during remodeling.
During the initial stages of wound healing, active fibroblasts rapidly deposit type III collagen, which forms a delicate, flexible framework within early granulation tissue. As healing progresses into the remodeling phase, this temporary type III collagen is enzymatically degraded and systematically replaced by type I collagen, which is structurally stronger and provides superior tensile strength.

179. (A) Directly performing the phagocytosis of invading bacteria
The extracellular matrix serves critical roles in tissue repair, including providing physical support for cell anchorage, acting as a reservoir for growth factors, and regulating cell proliferation via integrin signaling. However, the matrix is an acellular meshwork and cannot directly perform phagocytosis. Phagocytosis is a cellular function executed exclusively by specialized immune cells like macrophages and neutrophils.

180. (C) III and IV
Fibronectin is an extracellular adhesive glycoprotein rather than an intracellular protein, and it promotes cell migration by linking integrins to matrix components. Furthermore, the delicate granulation tissue of early wounds is rich in type III collagen, whereas type I collagen is deposited later during remodeling. Thus, the statements regarding fibronectin and type I collagen are incorrect.

NODIA

181. (B) Collagen cross-linking and tensile strength
Vitamin C is an essential cofactor required for the hydroxylation of proline and lysine residues during collagen synthesis. This enzymatic modification is critical for the formation of stable collagen triple helices. A severe vitamin C deficiency, known as scurvy, directly impairs collagen cross-linking, resulting in structurally weak fibers and compromised tensile strength in healing wounds.

182. (C) Degrade matrix to reshape the scar and clear temporary scaffolding.
Matrix metalloproteinases are zinc-dependent enzymes essential for the remodeling phase of wound healing. Their primary function is to degrade collagen and other extracellular matrix components in a highly controlled manner. This activity allows the body to reshape the scar, remove the temporary structural scaffolding, and replace weaker type III collagen with stronger type I fibers.

183. (D) Proteins (like fibronectin/laminin) connecting ECM elements and cells via integrins
Adhesive glycoproteins, such as fibronectin and laminin, are specialized proteins of the extracellular matrix. Their primary function is to act as molecular bridges that connect other matrix components, like collagen and proteoglycans, to cell surface receptors called integrins. This connection regulates key cellular processes, including survival, proliferation, and directed migration during active wound repair.

184. (A) Lysyl oxidases
Lysyl oxidases are extracellular copper-dependent enzymes responsible for the cross-linking of collagen fibrils during the maturation phase of tissue repair. These enzymes catalyze the oxidative deamination of specific lysine and hydroxylysine residues in collagen. This chemical reaction creates strong covalent bonds between adjacent fibrils, which substantially increases the tensile strength of the remodeling tissue scar.

185. (B) It promotes fibrosis by increasing collagen synthesis and decreasing MMP activity.
Transforming growth factor-beta is a major driver of tissue fibrosis. It upregulates the synthesis and deposition of extracellular matrix components, including collagen and fibronectin, by fibroblasts. Concurrently, it inhibits matrix degradation by downregulating matrix metalloproteinases and stimulating the production of tissue inhibitors of metalloproteinases, resulting in a net accumulation of dense fibrous scar tissue.

186. (B) It signals via intracellular cAMP instead of tyrosine kinase receptors.
Platelet-derived growth factor is stored in platelet alpha granules and is released upon platelet activation. It signals via cell surface receptor tyrosine kinases, rather than intracellular cyclic AMP. Once bound, it acts as a highly potent chemoattractant and mitogen that stimulates the migration and proliferation of fibroblasts, smooth muscle cells, and macrophages during tissue repair.

187. (A) Receptor tyrosine kinase activation, driving keratinocyte proliferation and migration
When epidermal growth factor binds to its specific receptor on basal keratinocytes, it triggers receptor homodimerization and autophosphorylation of its intracellular tyrosine kinase domain. This activates downstream intracellular signaling cascades, which drive keratinocyte mitotic proliferation and active migration across the wound surface. This process facilitates essential epidermal regeneration and rapid wound re-epithelialization in the skin.

188. (C) Vascular Endothelial Growth Factor (VEGF)
In managing a non-healing diabetic ulcer, applying recombinant vascular endothelial growth factor specifically targets and stimulates the process of angiogenesis. This growth factor binds to receptors on endothelial cells, promoting their proliferation and migration to form new blood vessels. This improves local perfusion and oxygenation, which are critical to overcome impaired healing of diabetic wounds.

189. (D) Receptor tyrosine kinases
Major growth factors involved in tissue repair, such as epidermal growth factor, vascular endothelial growth factor, and platelet-derived growth factor, signal through cell-surface receptor tyrosine kinases. Binding of the growth factor induces receptor dimerization and autophosphorylation of intracellular tyrosine residues. This initiates downstream signaling pathways that regulate cell proliferation, survival, and migration during wound healing.

Click Here to Buy book on Flipkart

190. (B) I, II and III
The vascular endothelial growth factor promotes angiogenesis, transforming growth factor-beta is a potent fibrogenic agent, and epidermal growth factor stimulates epithelial cell proliferation. In contrast, platelet-derived growth factor stimulates rather than inhibits fibroblast migration, and fibroblast growth factor promotes repair, meaning statements IV and V are incorrect while the first three statements are fully correct.

191. (B) Inhibiting extracellular matrix protein synthesis
Fibroblast growth factor is a multifunctional growth factor that plays vital roles in tissue repair. It promotes angiogenesis, stimulates the migration and proliferation of fibroblasts, and enhances epithelial cell migration to cover open epidermal wounds. It does not inhibit extracellular matrix protein synthesis; instead, it promotes the deposition and remodeling of matrix proteins during healing.

192. (D) Transforming Growth Factor-beta
Transforming growth factor-beta is a master regulatory cytokine that coordinates the transition from active inflammation to tissue repair and fibrosis. It acts to suppress the inflammatory response of leukocytes while powerfully stimulating fibroblasts to migrate, proliferate, and synthesize collagen. This makes it the key driver of tissue scarring and fibrosis, unlike classic pro-inflammatory systemic cytokines.

193. (A) EGF likely drives proliferation of various epithelial and mesenchymal cells during repair.
The widespread presence of epidermal growth factor receptors on various cellular populations beyond just keratinocytes indicates its broad physiological role. Epidermal growth factor acts as a highly potent mitogen that drives the proliferation, survival, and migration of many different epithelial and mesenchymal cell types, thereby coordinating complex cellular interactions required for efficient, coordinated tissue repair.

194. (C) It involves a smaller defect, less granulation, and minimal scarring.
Healing by primary intention occurs when clean wound edges are closely opposed, typically by surgical sutures. This clinical scenario minimizes the gap that must be filled, requiring less granulation tissue, fewer inflammatory cells, and minimal epithelial cell migration. Consequently, the healing process is rapid, efficient, and results in a significantly smaller, less noticeable fibrous scar.

195. (A) Both A and R are true and R is the correct explanation of A.
Wounds healing by secondary intention involve larger tissue defects where the edges cannot be approximated. These wounds require extensive granulation tissue to fill the large gap. Because of this volume, there is a much higher concentration of contractile myofibroblasts, leading to significant wound contraction compared to primary intention, making both the assertion and reason fully true.

196. (D) A clean, uninfected surgical incision closed by sutures
A clean, uninfected surgical incision closed by sutures represents the classical example of healing by primary intention. Because the wound edges are neatly and closely approximated, the defect is exceptionally small. This minimizes the need for extensive granulation tissue, limits the inflammatory response, and results in rapid epithelial closure with a minimal, thin fibrous scar.

197. (B) Abundant granulation tissue fills the large gap from the bottom up.
Healing by secondary intention occurs in wounds with large, unapproximated tissue defects. Because the wound edges are not opposed, the gap cannot be closed simply by epithelial migration. Instead, a large volume of active granulation tissue must form and fill the defect from the bottom up, which eventually leads to significant wound contraction and scarring.

NODIA

198. (C) Primary intention relies solely on epithelial regeneration with no connective tissue repair.
Healing by primary intention does not rely solely on epithelial regeneration; it also requires a coordinated connective tissue repair response. Although the defect is small, fibroblasts must still proliferate and synthesize collagen to physically unite the dermis. Both primary and secondary intention require an inflammatory phase and a fibroproliferative repair process to achieve successful final wound healing.

199. (D) Myofibroblasts forming a network that exerts mechanical pull on wound margins
Large surface ulcers healing by secondary intention exhibit intense wound contraction. This mechanical shrinkage is directly driven by specialized myofibroblasts, which express contractile α-smooth muscle actin. These cells proliferate within the granulation tissue and form a network that connects to the extracellular matrix, exerting a persistent, coordinated mechanical pull that draws the wound margins together.

200. (B) P-1, Q-2, R-3, S-4
Clean surgical incisions are approximated and heal by primary intention, resulting in minimal to no wound contraction. In contrast, large pressure ulcers feature extensive tissue loss and heal by secondary intention, requiring abundant granulation tissue and exhibiting significant wound contraction mediated by myofibroblasts. This maps each wound type directly to its healing intention and contraction characteristics.

201. (B) It requires extensive epidermal migration and proliferation over a large granulation bed.
In wounds healing by secondary intention, the process of epithelialization is much more extensive than in primary intention. Because there is a large, open tissue gap, the epidermal basal keratinocytes must undergo prolonged mitotic proliferation and migrate over a large, vascularized granulation tissue bed from the wound margins inward to completely cover the tissue defect.

202. (B) It grows beyond the original wound boundaries into healthy tissue.
A keloid is distinguished from a hypertrophic scar by its aggressive growth pattern. While a hypertrophic scar remains confined within the boundaries of the original wound and may spontaneously regress over time, a keloid grows beyond the original margins into surrounding healthy tissue and rarely regresses, resulting from excessive, disorganized collagen deposition in susceptible individuals.

203. (A) Adequate, robust vascular supply to the wound area
Numerous local factors can delay or compromise physiological wound healing, including localized infections, the presence of foreign bodies, and excessive mechanical motion or tension across the wound bed. In contrast, an adequate, robust vascular supply is highly beneficial, as it ensures the delivery of oxygen, nutrients, and immune cells necessary to support active tissue repair.

NODIA

204. (C) Excessive granulation tissue protruding above the skin, blocking re-epithelialization
Exuberant granulation, also known as proud flesh, is a pathological complication of wound healing. It is characterized by the overproduction of granulation tissue, which protrudes above the level of the surrounding skin. This physical protrusion acts as a mechanical barrier that completely blocks the migration of epithelial cells, thereby preventing successful re-epithelialization and wound closure.

205. (A) Both A and R are true and R is the correct explanation of A.
Wound dehiscence is the mechanical separation of previously approximated wound edges, most frequently observed in abdominal surgical incisions. These wounds are uniquely subjected to high mechanical tension and sudden increases in intra-abdominal pressure caused by coughing, vomiting, or abdominal distension. This mechanical stress can easily rupture the sutures, making both the assertion and reason fully true.

206. (C) It removes the physical barrier, letting epidermis migrate and close the defect.
Exuberant granulation tissue forms a prominent physical barrier that prevents re-epithelialization because the migrating epidermal cells cannot climb over the raised, protruding tissue. Surgical excision is therapeutically effective because it physically removes this excess tissue, restoring a flat wound bed. This allows the basal keratinocytes to migrate unimpeded across the surface to close the defect.

207. (B) The mechanical separation of previously approximated wound edges
Wound dehiscence is a significant clinical complication of tissue repair defined as the mechanical separation of previously approximated wound edges. This separation typically occurs in abdominal wounds subjected to increased physical tension or high pressure. It reflects an underlying weakness in collagen deposition, localized infection, or inadequate mechanical support from surgical sutures during healing.

208. (D) Wound contracture
Tight, disabling deformities around joints following severe burn injuries are classically recognized as wound contractures. This pathological complication arises when the physiological process of wound contraction is exaggerated. Driven by persistent, overactive myofibroblasts, the excessive shrinkage of scar tissue pulls surrounding structures inward, severely restricting joint mobility and causing permanent, severe physical deformities.

209. (A) Glucocorticoids impede healing by inhibiting inflammation and collagen synthesis.
Systemic factors can significantly impair the physiological wound healing process. Glucocorticoids are potent anti-inflammatory agents that impede tissue repair by suppressing the initial inflammatory response, which is essential to clear debris. Furthermore, they directly inhibit fibroblast activity and decrease collagen synthesis, leading to structurally weak scars. Other systemic impediments include protein malnutrition and uncontrolled hyperglycemia.

210. (C) P-2, Q-1, R-3, S-4
Pathological aberrations of wound healing exhibit distinct descriptive features. A keloid represents excessive scar tissue that grows beyond the boundaries of the original wound. A hypertrophic scar is a raised lesion that remains confined within the original borders. Proud flesh refers to excessive granulation tissue blocking epithelialization, and contractures are exaggerated shrinkages causing structural deformities.

****

Downloaded from www.pdf.tube

CHAPTER 04
Immunopathology

2024

1. What is the primary structural configuration of an immunoglobulin monomer?
(A) Four different heavy chains and two light chains without interchain bonds
(B) Two identical heavy chains and two identical light chains linked by disulfide bonds
(C) A single heavy chain covalently linked to multiple J-chains
(D) Two alpha chains and two beta chains connected by a secretory component

2004

2. Which of the following accurately describes the primary function of the hinge region in IgG and IgA molecules?
(A) It dictates the absolute half-life of the antibody in systemic circulation
(B) It binds directly to the complement C1q protein to initiate the classical pathway
(C) It anchors the immunoglobulin securely within the mucosal epithelial lining
(D) It provides segmental flexibility, allowing the two Fab arms to bind to separate antigenic determinants

2009

3. Which of the following statements concerning the immunoglobulin isotype IgM is INCORRECT?
(A) It requires interaction with the secretory component to function in intravascular spaces
(B) It is the first immunoglobulin synthesized by the neonate and during a primary immune response
(C) It exists primarily as a pentamer in serum, stabilized by a J-chain
(D) It is highly efficient at activating the classical complement pathway due to its multiple Fc regions

2010

4. Which of the following pairs correctly matches the immunoglobulin isotype with its primary biological function in animal health?
(A) IgD : Major mediator of Type I hypersensitivity in mucosal tissues
(B) IgE : Predominant antibody for systemic opsonization and placental transfer
(C) IgA : Primary secretory antibody protecting mucosal surfaces against enteric pathogens
(D) IgG : Exclusive membrane-bound receptor on mature, naive B-lymphocytes

2014

5. Which of the following pairs INCORRECTLY matches an immunoglobulin domain or chain with its specific immunological property?
(A) Secretory component : Drives somatic hypermutation
(B) Fab fragment : Determines specific antigen-binding site
(C) Fc fragment : Mediates binding to complement and phagocyte receptors
(D) J-chain : Facilitates polymerization of IgA and IgM

2014

6. All of the following are primary functions mediated by the Fc region of an IgG molecule EXCEPT:
(A) Complement activation via the classical pathway
(B) Facilitation of opsonization by binding to Fc-gamma receptors on macrophages
(C) Mediation of antibody-dependent cellular cytotoxicity (ADCC) by NK cells
(D) Binding to specific epitopes on pathogenic microorganisms

NODIA

2024

7. A mature B-lymphocyte typically co-expresses two distinct immunoglobulin isotypes on its surface as antigen receptors before activation. Which is the most appropriate pair?
(A) IgM and IgA
(B) IgM and IgD
(C) IgG and IgE
(D) IgA and IgE

2021

8. In a clinical scenario where a mucosal pathogen colonizes the respiratory tract of a calf, which isotype's polymeric structure provides the

highest avidity for viral neutralization at the mucosal surface?
(A) Monomeric IgG
(B) Monomeric IgE
(C) Dimeric IgA
(D) Monomeric IgD

2010

9. In the context of primary immune cells, excessive activation of helper T-lymphocytes invariably leads to the differentiation of naive B-lymphocytes into what cellular endpoint?
(A) Activated tissue-resident macrophages
(B) Cytotoxic $CD8^+$ T-lymphocytes
(C) Professional antigen-presenting dendritic cells
(D) Antibody-secreting plasma cells

2016

10. Arrange the following stages of B-lymphocyte development in the correct biological sequence:
I. Pre-B cell
II. Mature naive B cell
III. Pro-B cell
IV. Immature B cell
(A) I, III, II, IV
(B) III, I, IV, II
(C) III, IV, I, II
(D) I, IV, III, II

2017

11. How do Natural Killer (NK) cells accurately identify virus-infected host cells for destruction while sparing normal tissue cells?
(A) By recognizing viral peptides bound to host MHC class II molecules
(B) By detecting down-regulation of host MHC class I molecules
(C) By binding directly to free viral particles in extracellular fluids
(D) By utilizing surface receptors rearranged during thymic selection

2008

12. Which of the following represents an accurate comparison between conventional Dendritic Cells (cDCs) and Macrophages functioning as Antigen-Presenting Cells (APCs)?
(A) Macrophages are specialized for activating memory T cells, while cDCs primarily activate follicular B-lymphocytes
(B) cDCs present antigen in lymph nodes, while macrophages present to T cells in peripheral tissues
(C) Macrophages express only class I molecules, while cDCs express exclusively class II major histocompatibility molecules
(D) cDCs are derived from the lymphoid lineage, while macrophages derive from the erythroblast progenitor cells

2019

13. Which feature represents the least relevant characteristic of a fully differentiated effector $CD8^+$ T-lymphocyte during a viral infection?
(A) Direct somatic hypermutation of its T-cell receptor genes
(B) Secretion of perforin and granzymes to induce apoptosis
(C) High expression of Fas ligand (FasL) on the cell surface
(D) Production of interferon-gamma to enhance macrophage activation

2006

14. If a novel drug specifically blocks the CD28 receptor on naive T-lymphocytes, what can be logically inferred about the cellular immune response upon antigen encounter?
(A) T-lymphocytes will become fully activated purely through local TCR-MHC engagement
(B) Antigen-presenting cells will immediately undergo spontaneous receptor-mediated apoptosis
(C) The T-lymphocytes will enter a state of anergy due to the lack of costimulation
(D) B-lymphocytes will bypass T-cell help and undergo rapid direct class switching to IgE

2004

15. Consider the following statements regarding the functional characteristics of T-lymphocytes:
I. $CD4^+$ T cells recognize antigens presented by MHC class I molecules.
II. Th1 cells primarily secrete IFN-gamma and IL-2 to support cell-mediated immunity.
III. Regulatory T cells (Tregs) typically express CD4, CD25, and FoxP3.
Which of the above statement(s) is/are correct?
(A) I and II
(B) II and III
(C) I only
(D) I and III

2014

16. What is the defining advantage of cross-presentation by professional dendritic cells during an intracellular viral infection?
(A) It allows the direct transfer of intact viral proteins to follicular B cells to accelerate

Click Here to Buy book on Amazon

rapid systemic antibody production
(B) It triggers the immediate intracellular release of pre-formed neutralizing antibodies from active dendritic cell granules
(C) It down-regulates all surface MHC expression to completely protect the antigen-presenting cells from natural killer cell targeting
(D) It presents exogenous viral antigens via MHC class I to activate naive $CD8^+$ T cells without direct cell infection

2009
17. Which term defines a specialized primary lymphoid organ in birds where B-lymphocytes mature and differentiate?
(A) Peyer's patch
(B) Bone marrow
(C) Bursa of Fabricius
(D) Thymic medulla

2021
18. Upon recognizing an opsonized pathogen, which mechanism do macrophages primarily deploy to eliminate the threat?
(A) Secretion of intact immunoglobulins targeting the pathogen surface
(B) Phagocytosis followed by enzymatic degradation within the phagolysosome
(C) Spontaneous degranulation of histamine into the extracellular space
(D) Assembly of the membrane attack complex on the macrophage surface

2007
19. Mast cells, basophils, and eosinophils share which of the following definitive morphological and functional characteristics?
(A) They all mature exclusively in the thymus before migrating to the periphery
(B) They are the primary phagocytes responsible for clearing apoptotic host cells
(C) They predominantly express CD8 and kill target cells via perforin release
(D) They contain prominent cytoplasmic granules packed with inflammatory mediators

2006
20. Which of the following correctly identifies a primary effector mechanism of eosinophils when responding to large helminth parasites?
(A) Release of major basic protein and peroxidase onto the parasite's surface
(B) Intracellular digestion of the intact parasite inside giant phagolysosomes
(C) Presentation of helminth antigens via MHC class I directly to naive T cells
(D) Secretion of high-avidity IgM to neutralize parasite-derived toxic compounds

2011
21. Which statement INACCURATELY describes the physiological behavior of tissue mast cells?
(A) They are strategically distributed near blood vessels, nerves, and epithelial surfaces
(B) Their high-affinity surface receptors (FcεRI) bind circulating IgE even in the absence of antigen
(C) They circulate continuously in the blood and only enter tissues during active inflammation
(D) Upon activation, they synthesize leukotrienes and prostaglandins de novo from arachidonic acid

2023
22. Which pair correctly associates an effector cell with its prominent pre-formed primary mediator?
(A) Mast cell : Histamine
(B) Eosinophil : Immunoglobulin E
(C) Basophil : Granzyme B
(D) Macrophage : Serotonin

2006
23. Which pair INCORRECTLY matches the effector cell with its predominant localization in a healthy domestic animal?
(A) Mast cells : Connective tissue and mucosal layers
(B) Basophils : Peripheral blood circulation
(C) Macrophages : Distributed widely across peripheral tissues
(D) Eosinophils : Exclusively confined to primary lymphoid organs

NODIA

2011
24. All of the following mediators are rapidly secreted by mast cells following antigen-induced degranulation EXCEPT:
(A) Histamine
(B) Major Basic Protein (MBP)
(C) Heparin
(D) Serotonin

2017
25. Consider the following statements concerning the biology of basophils and mast cells:
I. Both cells express high-affinity Fc receptors for IgE (FcεRI).
II. Basophils are primarily tissue-resident cells, while mast cells circulate in the blood.
III. Both release histamine upon cross-linking of surface-bound IgE.

2003

55. Match the hypersensitivity types in List I with their defining immunological mediators in List II according to the Gell and Coombs classification:

List I: (Hypersensitivity Type)

P. Type I
Q. Type II
R. Type III
S. Type IV

List II: (Immunological Mediator)

1. Soluble circulating immune complexes
2. T-lymphocytes and macrophages
3. IgE and mast cells
4. IgG or IgM directed against cell surface antigens

(A) P-1, Q-4, R-3, S-2
(B) P-3, Q-4, R-1, S-2
(C) P-3, Q-1, R-4, S-2
(D) P-2, Q-3, R-1, S-4

2023

56. Which of the following is considered an INCORRECT pair based on the Gell and Coombs hypersensitivity classification system?

(A) Type I : Anaphylactic or immediate hypersensitivity
(B) Type II : Cytotoxic or cell-stimulating hypersensitivity
(C) Type III : Immune complex-mediated hypersensitivity
(D) Type IV : IgA-dependent mucosal hypersensitivity

2005

57. The primary pathophysiological mechanism distinguishing Type III from Type II hypersensitivity relates to which of the following characteristics?

(A) Type III involves antibodies targeting fixed tissue antigens in organs, whereas Type II involves circulating soluble antigens
(B) Type III utilizes cytotoxic $CD8^+$ T cells exclusively during the effector stage, whereas Type II utilizes classical B cells
(C) Type III involves deposition of circulating complexes, while Type II involves antibodies binding directly to cell surfaces
(D) Type III is a rapid, immediate mast-cell dependent localized reaction, whereas Type II typically takes several weeks to develop

2005

58. All of the following cellular components or molecules are integral to the pathogenesis of Type IV hypersensitivity EXCEPT:

(A) $CD4^+$ Th1 lymphocytes
(B) Macrophages
(C) Interferon-gamma (IFN-γ)
(D) Histamine-laden basophils

2010

59. When categorizing a delayed skin reaction such as the tuberculin test, which specific mechanism correctly explains the 48-to-72 hour delay in the observed swelling?

(A) It requires time for antigen processing, T-cell activation, and the subsequent recruitment of macrophages
(B) It takes this amount of time for circulating pre-formed IgE antibodies to cross-link on tissue mast cells
(C) It takes 72 hours for the alternative complement pathway to initiate spontaneous fluid-phase C3 cleavage
(D) It requires this specific duration of time for maternal antibodies to be fully cleared from the system

2021

60. What is the fundamental concept underlying the pathogenesis of Type I hypersensitivity (Anaphylaxis)?

(A) Degranulation of basophils triggered by complement activation
(B) Cross-linking of mast cell-bound IgE by a multivalent allergen
(C) Phagocytosis of harmless environmental allergens by neutrophils
(D) Formation of massive IgG complexes that occlude capillaries

2021

61. Which sequence appropriately details the events leading to sensitization in Type I hypersensitivity?

(A) Allergen exposure → Th1 activation → IFN-gamma release → Macrophage activation → Histamine release
(B) Allergen exposure → Th2 activation → IL-4 production → B-cell isotype switch → IgE binding to mast cells
(C) IgE binds to mast cells → Allergen exposure → Th2 activation → IL-4 production → B-cell apoptosis
(D) Mast cell degranulation → Allergen exposure → IgE synthesis → Th2 activation → IL-4 release

Downloaded from www.pdf.tube

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

(C) The C5a fragment acts as a potent anaphylatoxin and neutrophil chemoattractant
(D) The C5b fragment acts as a standalone protease that degrades bacterial peptideglycan

2021

34. Which of the following defines the primary enzymatic function of the C5 convertase complex?
(A) Cleavage of intact C3 molecules into active fragments C3a and C3b
(B) Cleavage of C5 into C5a and C5b to initiate the terminal pathway
(C) Polymerization of multiple C9 molecules into a transmembrane channel
(D) Hydrolysis and inactivation of antigen-bound circulating immunoglobulins

2005

35. Consider the following statements concerning the terminal complement pathway and cellular lysis:
I. The terminal pathway is identical regardless of the initial activation route (classical, alternative, or lectin).
II. C5b interacts with C6, C7, and C8 to form a complex that inserts into the cell membrane.
III. C3a is the primary structural component of the Membrane Attack Complex pore.
IV. Poly-C9 assembly completes the structural pore, leading to osmotic lysis.
V. Host cells lack mechanisms to defend against accidental MAC formation.
Which of the above statement(s) is/are correct?
(A) I, II, III and V
(B) I, II, IV and V
(C) I, II and IV
(D) II, IV and V

2013

36. Which milestone reflects the earliest functional capability of the mammalian fetal immune system during ontogeny?
(A) Appearance of early lymphoid organs and lymphocyte precursors
(B) Secretion of high-avidity, class-switched antigen-specific IgA
(C) Generation of mature, antigen-specific memory helper T cells
(D) Development of robust Type IV delayed hypersensitivity responses

2001

37. Which statement represents an accurate timeline concept in the immune-competence development of a mammalian fetus?
(A) The fetus relies entirely on passive colostrum before birth and fails to develop any functional immune structures in utero
(B) Humoral immunity matures completely and achieves full antigen-responsiveness before cellular immunity begins to develop
(C) Immunocompetence develops in a stepwise manner, recognizing different antigens at progressive gestational stages
(D) Fetuses cannot produce any protective type-I or type-II interferons until several weeks following post-parturition

2007

38. Which of the following statements about the ontogeny of the immune system is INCORRECT?
(A) Maternal IgG completely suppresses fetal hematopoiesis until after birth
(B) The thymus is generally the first lymphoid organ to develop in the mammalian fetus
(C) Circulating fetal B-lymphocytes predominantly express IgM and IgD as surface receptors
(D) Fetal lymphocytes can mount an immune response to certain viral pathogens if infected in utero

NODIA

2014

39. Which pair correctly matches the developmental stage of the immune system with its functional hallmark?
(A) Mid-gestation fetal liver : Exclusive anatomical site of memory B cell storage
(B) Late-gestation thymus : Primary microenvironment for systemic antibody production
(C) Post-natal spleen : Initial anatomical site of T cell receptor gene rearrangement
(D) Early embryonic yolk sac : Site of initial hemopoiesis and stem cell generation

2001

40. Which pair INCORRECTLY associates an aspect of fetal immunity with its physiological reality?
(A) Fetal viral infection : May result in tolerance if exposed before full immunocompetence
(B) Fetal antibody production : Heavily skewed toward the production of IgE
(C) Bovine viral diarrhea virus (BVDV) exposure : Can cause persistent infection if it occurs

IV. Mast cells play a critical role in the pathogenesis of Type I hypersensitivity.

Which of the above statement(s) is/are correct?

(A) I, II and IV
(B) I, III and IV
(C) II and III
(D) I and IV

2017

26. In an allergic inflammatory response, the localized release of histamine by mast cells leads to which of the following immediate physiological effects?

(A) Systemic down-regulation of bone marrow hematopoiesis
(B) Immediate induction of B-cell isotype switching to IgG
(C) Rapid apoptosis of adjacent regulatory T cells
(D) Vasodilation and increased vascular permeability

2003

27. The classical pathway of the complement system is most efficiently initiated by which specific intermolecular interaction?

(A) Binding of the C1 complex to antigen-bound IgG or IgM molecules
(B) Spontaneous fluid-phase hydrolysis of C3 molecules in the plasma
(C) Binding of mannose-binding lectin to pathogen surface carbohydrates
(D) Direct binding of properdin to the surface of Gram-negative bacteria

2014

28. Which of the following classifications appropriately describes the active C3 convertase of the alternative complement pathway?

(A) C4b2a
(B) C3bBb
(C) C1qrs
(D) C5b678

2005

29. Compare the alternative and classical complement pathways regarding their reliance on adaptive immunity. Which statement is accurate?

(A) Both pathways require pre-existing antigen-specific antibodies bound to surface epitopes for optimal enzymatic activation
(B) The classical pathway is antibody-dependent, while the alternative pathway functions as an antibody-independent innate defense
(C) The alternative pathway depends heavily on $CD8^+$ T-lymphocyte cell activation, whereas the classical pathway relies on helper B cells
(D) Both pathways bypass C3 cleavage and directly activate the terminal membrane attack complex in the absolute absence of antibodies

2009

30. What is the least relevant outcome of complement pathway activation during an acute bacterial infection?

(A) Induction of specific somatic hypermutation in memory B cells
(B) Assembly of the Membrane Attack Complex (MAC) for bacterial lysis
(C) Opsonization of the pathogen via C3b binding
(D) Chemotactic recruitment of neutrophils via C5a

2001

31. If an animal has a genetic deficiency preventing the synthesis of the C9 complement protein, which specific immunological defect can be reliably inferred?

(A) Total failure to initiate the classical pathway upon antigen exposure
(B) Complete inability to assemble a functional C3 convertase complex
(C) Defective assembly of the cytolytic Membrane Attack Complex (MAC) pore
(D) Complete absence of neutrophil chemotaxis to localized infection sites

2014

32. In the context of complement activation, which of the following represents an elimination mechanism to prevent uncontrolled host tissue damage?

(A) Continuous up-regulation of active properdin on all healthy host cell membranes
(B) Rapid secretion of C5a molecules to down-regulate local tissue macrophage activity
(C) Substitution of C3 glycoproteins with immunoglobulins in the alternative pathway
(D) Expression of CD59 and Decay Accelerating Factor (DAF) on host membranes

2015

33. The cleavage of C5 during complement activation exerts which of the following direct effects?

(A) The C5b fragment competitively inhibits the action of C3 convertase
(B) The C5a fragment binds directly to mast cell Fc receptors to inhibit degranulation

Click Here to Buy book on Flipkart

whereas pigs transfer virtually no IgG prenatally
(D) Pigs transfer IgG via an endotheliochorial mechanism, while humans lack active transport entirely

2023
48. What is the least relevant factor when evaluating the mechanism of maternal antibody transfer across a hemochorial placenta (e.g., in primates)?
(A) Expression of the neonatal Fc receptor (FcRn) on the syncytiotrophoblast
(B) The isotype of the antibody, specifically the binding affinity of the IgG Fc region
(C) The high concentration of circulating IgG in the maternal plasma
(D) The direct mixing of maternal and fetal erythrocytes in the chorionic villi

2002
49. Based on placental anatomy, which outcome logically follows for a neonatal calf that is deprived of colostrum immediately after birth?
(A) It will rely entirely on its naturally high reservoir of transplacental maternal IgG
(B) It will immediately synthesize adult-level protective IgA to compensate for the lack
(C) It will suffer from agammaglobulinemia and be highly susceptible to septicemia
(D) It will exhibit spontaneous, acute autoimmune hemolytic anemia in systemic tissues

2001
50. By what primary mechanism does the neonatal enterocyte facilitate the absorption of colostral immunoglobulins during the first 24 hours of life?
(A) Active transport coupled directly and exclusively to sodium-potassium ATPase
(B) Non-selective fluid-phase pinocytosis followed by systemic transcytosis
(C) Specific carrier-mediated facilitated diffusion of monomeric amino acids
(D) Direct passive diffusion of IgG through tight junctions between enterocytes

2023
51. What is the major physiological advantage of the "gut closure" phenomenon that occurs in neonates roughly 24 hours post-partum?
(A) It permanently prevents the systemic mucosal absorption of toxic environmental heavy metals
(B) It triggers the complete structural remodeling of the stomach into four distinct compartments
(C) It actively initiates the hepatic synthesis and secretion of maternal bile salts into the lumen
(D) It stops the unselective uptake of large macromolecules and potential intestinal pathogens

2015
52. Which of the following is an INCORRECT statement regarding the pathophysiology of Failure of Passive Transfer (FPT)?
(A) FPT induces a primary genetic mutation in the neonate's B-lymphocytes, preventing future antibody synthesis
(B) FPT can occur even if the dam produces adequate colostrum but the neonate is too weak to nurse
(C) Premature lactation in the mare leads to a loss of high-quality colostrum before the foal is born, causing FPT
(D) Delayed feeding of colostrum past 24 hours inevitably leads to FPT due to intestinal closure

2021
53. If a foal is diagnosed with Failure of Passive Transfer (FPT) at 36 hours of age, which intervention represents the most appropriate cause-and-effect clinical response?
(A) Immediate oral administration of bovine colostrum because the foal's gut remains highly permeable
(B) Intramuscular injection of killed viral vaccines to instantly stimulate the foal's innate immunity
(C) Intravenous administration of hyperimmune plasma because the gut is closed to oral immunoglobulins
(D) Strict isolation without medical treatment, relying entirely on endogenous antibody production

2001
54. Which pair correctly relates a parameter to its established diagnostic threshold for adequate passive transfer of immunity in a neonatal foal?
(A) Serum IgG $>$ 800 mg/dL : Adequate passive transfer
(B) Serum IgM $<$ 100 mg/dL : Partial failure of passive transfer
(C) Colostral specific gravity $<$ 1.020 : Excellent colostrum quality
(D) Total serum protein $<$ 3.5 g/dL : Complete protection against sepsis

Downloaded from www.pdf.tube

(C) Natural Killer (NK) cell
(D) Mature erythrocyte

2008

70. Neonatal isoerythrolysis in foals is a classic veterinary example of which underlying pathological mechanism?
(A) A Type I immediate allergic response to nutritive proteins present in maternal colostrum
(B) A Type II reaction where maternal colostral alloantibodies destroy foal red cells
(C) A Type III reaction where circulating immune complexes deposit in the fetal kidney
(D) A Type IV cell-mediated rejection of active maternal lymphocytes by foal T cells

2003

71. Which condition correctly exemplifies a drug-induced cytopenia operating via a Type II hypersensitivity mechanism?
(A) Administration of penicillin leading directly to acute systemic anaphylactic shock
(B) A drug directly poisoning bone marrow hemopoietic stem cells via toxic metabolites
(C) A drug inducing an intense, chronic localized granuloma at the dermal injection site
(D) A drug hapten binding red cells and eliciting an IgG response that destroys them

2013

72. During an incompatible blood transfusion reaction, the primary cause of acute intravascular hemolysis is:
(A) The massive, immediate release of vasoactive histamine from basophils attacking the donor blood cells
(B) Recipient antibodies binding donor RBC membranes to activate the classical complement cascade
(C) The direct activation of specific cytotoxic T-cells that perforate and lyse the donor RBCs
(D) The rapid mechanical shearing of circulating RBCs by newly formed microthrombi in vessels

2015

73. Which of the following is a negative outcome directly linked to the excessive opsonization of red blood cells by IgG and C3b in autoimmune hemolytic anemia?
(A) Enhanced physiological survival of coated RBCs in the peripheral circulation
(B) Rapid synthesis of new RBCs by the bone marrow to completely exceed destruction
(C) Accelerated splenic macrophage phagocytosis of RBCs (extravascular hemolysis)
(D) Immediate down-regulation of all classical and alternative complement pathways

2003

74. If a severe mismatched blood transfusion occurs in a previously sensitized dog, what is the most appropriate deduction regarding the immediate pathophysiological events?
(A) The dog will mount a delayed Type IV cell-mediated response against the foreign blood type over the next week
(B) The transfused donor cells will undergo immediate apoptosis due to slight changes in physiological temperature
(C) The dog's regulatory T cells will actively suppress all localized responses to prevent host damage
(D) Pre-existing IgG or IgM will bind transfused erythrocytes, triggering complement activation and hemolysis

NODIA

2006

75. Given below are two statements: one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): In immune-mediated hemolytic anemia (IMHA), the direct Coombs test is a critical diagnostic tool.
Reason (R): The direct Coombs test detects the presence of host immunoglobulins or complement proteins bound to the surface of the patient's washed red blood cells.
In the light of the above statements, choose the most appropriate answer from the options given below:
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2004

76. By what primary mechanism does the deposition of soluble immune complexes in Type III hypersensitivity initiate severe tissue damage?
(A) By directly inducing target apoptosis of endothelial cells via localized surface Fas-FasL interactions
(B) By activating complement to generate C5a, which recruits neutrophils that release

2010

62. Which of the following represents the least relevant factor in determining an individual animal's propensity to develop atopic dermatitis?
(A) The individual's blood type and erythrocyte surface antigens
(B) Genetic predisposition to produce high levels of IgE (atopy)
(C) The presence of specific environmental allergens
(D) Epidermal barrier dysfunction increasing allergen penetration

2014

63. During a systemic anaphylactic shock event, the rapid release of secondary lipid mediators such as leukotrienes and prostaglandins primarily exerts which effect?
(A) Rapid acceleration of blood coagulation resulting in disseminated intravascular coagulation (DIC)
(B) Immediate destruction of circulating erythrocytes
(C) Profound, sustained bronchoconstriction and widespread vasodilation causing hypotension
(D) Inhibition of mucus production in the respiratory tract

2025

64. A dog is brought to the clinic with severe facial swelling, erythema, and pruritus 20 minutes after a bee sting. This presentation is most accurately classified as which type of immune-mediated reaction?
(A) Type III localized Arthus reaction
(B) Type II cytotoxic immune response
(C) Type I immediate hypersensitivity reaction
(D) Type IV delayed cell-mediated response

2021

65. Choose the correct sequence of events in the localized Type III hypersensitivity Arthus Reaction from the options provided.
(A) Allergen exposure → IgE cross-linking on mast cells → Massive degranulation → Localized cutaneous edema
(B) T cell activation → IFN-gamma release → Chemotactic macrophage recruitment → Chronic granuloma formation
(C) Antigen injection → IgG complex formation → Complement activation → Neutrophil infiltration and necrosis
(D) RBC antigen mismatch → Recipient IgG binding → Classical complement activation → Acute intravascular hemolysis

2006

66. Which statement accurately compares systemic anaphylaxis and atopy?
(A) Systemic anaphylaxis is a localized Type IV cellular response, whereas atopy is a systemic Type III complex response
(B) Both are Type I reactions, but anaphylaxis involves systemic shock, while atopy is localized or regional
(C) Atopy is mediated primarily by IgG antibodies, whereas systemic anaphylaxis is exclusively mediated by IgM
(D) Atopy requires classical complement activation for tissue damage, whereas systemic anaphylaxis does not

2009

67. Which of the following is an INCORRECT statement regarding the clinical veterinary manifestations of allergic respiratory diseases, such as Feline Asthma or Equine Asthma (Heaves)?
(A) They involve chronic airway inflammation frequently dominated by eosinophils and mast cells
(B) Clinical signs include bronchospasm, increased mucus production, and airway remodeling
(C) Environmental management (e.g., reducing dust and molds) is a key component of mitigating the hypersensitivity response
(D) The pathogenesis is strictly limited to Type II cytotoxic hypersensitivity destroying the respiratory epithelium

2011

68. The destruction of host target cells in Type II hypersensitivity relies primarily on which effector mechanisms?
(A) Complement lysis, antibody-dependent cellular cytotoxicity, and opsonization
(B) Th1 cell-mediated target cell apoptosis and classical macrophage activation
(C) Allergen-induced IgE cross-linking followed by rapid mast cell degranulation
(D) Deposition of small immune complexes in filtering membranes like the glomerulus

2025

69. In antibody-dependent cellular cytotoxicity (ADCC) associated with Type II hypersensitivity, which cell type acts as the principal effector utilizing its Fcγ receptors to bind the target?
(A) B-lymphocyte
(B) Cytotoxic $CD8^+$ T-lymphocyte

Click Here to Buy book on Amazon

early in gestation
(D) Neonatal cellular immunity : Functional but generally naive and slower to respond than an adult's

2006
41. All of the following factors contribute to the relative immunodeficiency observed in normal mammalian neonates EXCEPT:
(A) High levels of endogenous circulating corticosteroids related to birth stress
(B) Naive status of both T and B lymphocyte populations
(C) Complete anatomical absence of secondary lymphoid organs like lymph nodes
(D) Down-regulated APC functions, including lower MHC class II expression

2025
42. In a newborn foal, what is the most appropriate indicator that the fetus mounted an active immune response to an in utero infection rather than acquiring antibodies passively?
(A) High levels of IgG in the foal's serum immediately after nursing
(B) High levels of maternal IgA in the fetal circulation
(C) Rapid development of a robust contact dermatitis to maternal colostrum
(D) Presence of elevated specific IgM antibodies in the pre-suckle serum

2017
43. Applying concepts of fetal immune development, why does early gestation exposure to non-cytopathic BVDV in calves lead to a persistently infected, immunotolerant state?
(A) Viral antigens in the thymus trigger clonal deletion of specific T-cells during negative selection
(B) The virus genetically deletes the MHC class I locus in the bovine host genome
(C) Maternal antibodies actively target and destroy all fetal lymphocyte populations that recognize the virus
(D) The fetus exclusively produces non-neutralizing IgE antibodies which are quickly degraded and depleted

2014
44. Infection of a mammalian fetus with a specific pathogen typically results in which sequence of pathological or immunological effects depending on the gestational age?
(A) Robust neutralizing antibody response if early, strict tolerance if late
(B) Mummification/abortion if early, immune clearance/survival if late
(C) Immediate induction of autoimmune disease regardless of timing
(D) Severe combined immunodeficiency (SCID) regardless of timing

2019
45. Given below are two statements: one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): In domestic ungulates such as horses and cattle, the transfer of maternal immunoglobulins to the fetus via the placenta is negligible.
Reason (R): These species possess an epitheliochorial placenta, which features multiple tissue layers separating the maternal and fetal bloodstreams, blocking large molecular transfer.
In the light of the above statements, choose the most appropriate answer from the options given below:
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2010
46. Which classification correctly categorizes the placental type of dogs and cats regarding its efficiency in maternal antibody transfer?
(A) Epitheliochorial placenta preventing transplacental immunoglobulin transfer entirely
(B) Endotheliochorial placenta allowing partial transfer (5-10%) of maternal IgG
(C) Hemochorial placenta allowing complete transplacental transfer of maternal IgG
(D) Syndesmochorial placenta restricting transfer completely to passive mucosal IgA

NODIA

2025
47. Compare the placental transfer of immunity in humans versus pigs. Which statement is accurate?
(A) Humans rely entirely on colostral antibody transfer, while pigs rely on active transplacental transfer
(B) Both species rely equally on highly efficient transplacental transfer of protective IgM and IgA
(C) Humans transfer IgG across the placenta,

destructive enzymes
(C) By cross-linking allergen-specific IgE on local tissue mast cells, causing mild and transient edema
(D) By blocking the total oxygen binding capacity of local circulating red blood cells, causing hypoxia

2002
77. Which statement represents an INCORRECT pairing of a disease with its corresponding hypersensitivity classification?
(A) Serum sickness : Type III hypersensitivity
(B) Contact dermatitis to poison ivy : Type II hypersensitivity
(C) Immune-mediated glomerulonephritis : Type III hypersensitivity
(D) Systemic Lupus Erythematosus (vascular lesions) : Type III hypersensitivity

2003
78. In the context of Type III hypersensitivity, what is the primary determinant causing soluble immune complexes to deposit within vascular walls or filtering tissues rather than being cleared from circulation?
(A) The complexes form at intermediate antigen-antibody ratios, preventing efficient phagocytic clearance
(B) The complexes are purely composed of IgM molecules and are too large for standard macrophage recognition
(C) The complexes completely lack any bound complement proteins, masking them from tissue phagocytes
(D) The complexes are actively transported into peripheral tissues by specific mature B-lymphocytes

2008
79. Which of the following is a classic clinical manifestation of localized Type III hypersensitivity in the eye of a dog?
(A) Retinal degeneration driven by local cytotoxic T cells
(B) Cataract formation due to excessive aqueous humor IgE
(C) Adenovirus-induced "blue eye" from complex deposition
(D) Mast cell tumor formation on the palpebral margin

2004
80. Identify the INCORRECT statement regarding the pathogenesis of immune-mediated glomerulonephritis (a Type III disease).
(A) Circulating immune complexes become trapped in the glomerular basement membrane during plasma filtration
(B) Trapped complexes activate complement, attracting neutrophils that damage the glomerulus
(C) Over time, the chronic inflammation causes thickening of the basement membrane and loss of protein (proteinuria)
(D) The damage invariably leads to massive polyuria and immediate acute renal failure within minutes

2003
81. Serum sickness was historically observed following the administration of large volumes of foreign antitoxin serum (e.g., equine tetanus antitoxin given to humans). What is the physiological sequence of events?
(A) Immediate IgE cross-linking on tissue-resident mast cells, leading to systemic anaphylactic shock within 10 minutes of administration
(B) Direct biochemical lysis of host vascular endothelial cells by highly reactive enzymatic foreign equine proteins in serum
(C) Antibody formation against foreign serum over 7-10 days, leading to complexes, complement activation, and vasculitis
(D) A pure, delayed cell-mediated immune rejection of foreign proteins by cytotoxic $CD8^+$ T cells over a span of 4 weeks

2007
82. Which of the following represents the least relevant finding in a joint fluid aspirate from an animal suffering from immune-mediated polyarthritis (a Type III hypersensitivity)?
(A) Presence of high numbers of non-degenerate neutrophils in fluid
(B) Presence of intracellular bacteria undergoing binary fission
(C) Decreased viscosity of synovial fluid due to enzymatic degradation
(D) Localized consumption of classical and alternative complement

2002
83. The clinical manifestation of Type IV delayed hypersensitivity fundamentally relies upon the

Downloaded from www.pdf.tube

action of which specific cytokine to recruit and highly activate tissue macrophages?
(A) Interleukin-4 (IL-4)
(B) Transforming Growth Factor-beta (TGF-β)
(C) Interleukin-10 (IL-10)
(D) Interferon-gamma (IFN-γ)

2003
84. In the comparative pathogenesis of delayed hypersensitivity, why is the tuberculin test read at 72 hours rather than 1 hour post-injection?
(A) Memory Th1 cells must encounter the antigen, secrete active cytokines, and recruit macrophages, which is a slow physiological process
(B) The injected tuberculin glycoprotein requires a full 72 hours to structurally alter and transition into its immunogenic form
(C) It takes 72 hours for the host immune system to synthesize entirely new classes of antigen-specific antibodies from naive B cells
(D) The local inflammatory reaction occurs instantly, but observable tissue swelling is physically delayed by high bovine dermal density

2016
85. Consider the contact dermatitis caused by chemicals (such as flea collar resins or topical drugs):
I. It is a form of Type IV hypersensitivity.
II. The offending chemical acts as a hapten, binding to host skin proteins to become immunogenic.
III. The reaction is driven by the immediate release of histamine from mast cells.
Which of the above statement(s) is/are correct?
(A) I and III
(B) I and II
(C) II and III
(D) I, II and III

2014
86. When an intracellular pathogen, such as Mycobacterium bovis, resists destruction within a macrophage, how does the Type IV hypersensitivity mechanism attempt to contain the infection?
(A) By driving the formation of a chronic granuloma composed of macrophages, giant cells, and lymphocytes
(B) By synthesizing large amounts of antigen-specific secretory IgA to neutralize the intracellular bacteria
(C) By inducing acute systemic anaphylaxis and vascular changes to actively flush the bacteria from tissues
(D) By activating the alternative complement pathway to directly lyse the infected host macrophage from the outside

NODIA

2019
87. Which elimination mechanism prevents normal, healthy domestic animals from mounting an immune response against their own physiological tissues?
(A) Spontaneous somatic mutation of self-antigens to avoid immune detection
(B) Continuous suppression of all B and T cell activity in peripheral blood
(C) Absolute lack of functional MHC class I molecules on healthy host cells
(D) Induction of central and peripheral immunological tolerance to self-antigens

2010
88. How does central tolerance mechanically act within the thymus to prevent autoimmunity?
(A) By actively mutating T-cell receptor genes of autoreactive clones until they no longer recognize host proteins
(B) By triggering apoptosis (negative selection) in developing T-cells that bind self-antigens in the thymus
(C) By isolating all autoreactive T-lymphocytes inside a specialized, completely impermeable thymic granuloma
(D) By directing newly generated autoreactive T-cells to secrete immunosuppressive IgG antibody variants

2013
89. Which concept explains the breakdown of self-tolerance through "molecular mimicry"?
(A) Host cells randomly mimic the outer morphology of foreign pathogens, tricking the immune system into attacking them
(B) Regulatory T cells spontaneously adopt the destructive phenotype of cytotoxic T cells and destroy normal tissues
(C) A pathogen antigen resembles a host self-antigen, causing the immune response to cross-react with host tissues
(D) B-lymphocytes synthesize highly non-specific antibodies that stick randomly and non-selectively to all cell surfaces

2018
90. The exposure of "cryptic epitopes" can lead to the breakdown of peripheral tolerance and

subsequent autoimmune disease. What is the cause-and-effect mechanism behind this?

(A) The host cells genetically insert foreign viral DNA directly into their own MHC loci, completely hiding their cellular identity
(B) Active tissue macrophages continuously hide foreign antigens inside intracellular lipid vacuoles to prevent adaptive immune detection
(C) Mature T cells down-regulate their TCRs to become functionally "cryptic," subsequently attacking healthy peripheral tissues blindly
(D) Inflammation or injury alters self-proteins, revealing hidden antigenic determinants to which the system was not tolerized

2025

91. In the context of autoimmune diseases, "epitope spreading" implies which of the following progressive changes in the immune response?

(A) An initial immune response broadens to target distinct epitopes on the same or related autoantigens as damage continues
(B) The rapid systemic spread of a single viral epitope to multiple different host organs and vascular beds via the bloodstream
(C) The rapid somatic mutation of a single active B-cell clone to produce identical specificity antibodies across all isotypes
(D) The deliberate therapeutic administration of multiple different foreign antigens over time to induce peripheral tolerance

2024

92. Systemic Lupus Erythematosus (SLE) in dogs is characterized primarily by which underlying immunological defect?

(A) A severe primary congenital immunodeficiency characterized by a total lack of functional mature T-lymphocytes
(B) A breakdown in tolerance producing autoantibodies, like ANA, causing systemic immune complex deposition
(C) The spontaneous systemic degranulation of tissue-resident mast cells causing chronic, low-grade anaphylaxis
(D) An absolute genetic failure of the bone marrow progenitor cells to produce any functional circulating neutrophils

2007

93. Which of the following defines the classical "LE cell" phenomenon associated with Systemic Lupus Erythematosus?

(A) A cytotoxic T cell that has absorbed large quantities of circulating autoantibodies onto its surface receptors
(B) A phagocyte that has ingested nuclear material previously opsonized by antinuclear antibodies from circulating serum
(C) A mature tissue macrophage that has failed to completely digest a phagocytized pathogenic mycobacterial organism
(D) A specifically mutated B-lymphocyte that permanently resides in dermal tissue, causing active lupus skin lesions

2003

94. The clinicopathological features of canine SLE frequently include polyarthritis, glomerulonephritis, and dermatological lesions. These specific signs are most directly the consequence of:

(A) The vascular deposition of circulating immune complexes followed by complement-mediated inflammation
(B) Direct infiltration of the affected tissues by malignant plasma cells
(C) Severe secondary viral infections due to viral-induced immunosuppression
(D) The continuous mechanical trauma of normal host movements without proper synovial fluid

2004

95. Consider the Pemphigus complex of organ-specific autoimmune diseases:

I. It is characterized by autoantibodies targeting desmosomal proteins in the epidermis.
II. The antibody binding leads to acantholysis (loss of cohesion between keratinocytes).
III. Pemphigus foliaceus targets deeper epidermal layers than Pemphigus vulgaris.
IV. Clinical signs typically include vesicles, pustules, and crusting of the skin or mucocutaneous junctions.

Which of the above statement(s) is/are correct?

(A) I, II and IV
(B) I, II and III
(C) II, III and IV
(D) I, III and IV

2011

96. Which organ-specific autoimmune disease is accurately linked to its targeted autoantigen causing failure of neuromuscular transmission?

(A) Autoimmune hemolytic anemia : Erythrocyte Band 3 membrane protein
(B) Pemphigus vulgaris : Epidermal cell-surface

Click Here to Buy book on Flipkart

P. Cytotoxic T cell
Q. Helper T cell
R. Regulatory T cell
S. B cell

List II: (Primary Role/Defining Marker)

1. $CD4^+$ and secretes cytokines to direct the immune response
2. $CD8^+$ and directly kills infected host cells
3. Fox $P3^+$ and suppresses excessive immune reactions
4. Expresses surface immunoglobulin and differentiates into plasma cells

(A) P-1, Q-2, R-3, S-4
(B) P-2, Q-3, R-1, S-4
(C) P-3, Q-1, R-4, S-2
(D) P-2, Q-1, R-3, S-4

2011

126. An animal with a severe deficiency in the production of Interleukin-2 (IL-2) would experience a profound failure in which of the following processes?
(A) Secretion of stomach acid by parietal cells
(B) Immediate degranulation of basophils in the blood
(C) Clonal expansion and proliferation of activated T-lymphocytes
(D) The physical transfer of antibodies across the placental barrier

2015

127. Which pair correctly identifies an autoimmune condition with its primary hypersensitivity mechanism?
(A) Systemic Lupus Erythematosus : Type II Hypersensitivity
(B) Autoimmune Hemolytic Anemia : Type II Hypersensitivity
(C) Myasthenia Gravis : Type I Hypersensitivity
(D) Pemphigus Foliaceus : Type III Hypersensitivity

2010

128. What is the most appropriate term for an antigen that causes an allergic Type I hypersensitivity reaction?
(A) Superantigen
(B) Hapten
(C) Tolerogen
(D) Allergen

2011

129. By what mechanism does the tuberculin skin test (purified protein derivative - PPD) reliably identify an animal previously infected with Mycobacterium bovis?
(A) It elicits a Th1 response, releasing cytokines that recruit macrophages and cause induration
(B) It triggers pre-formed IgE, causing immediate wheal and flare formation within 15 minutes
(C) It reacts with circulating IgG to form local immune complexes and Arthus-type tissue necrosis
(D) It causes an immediate, massive influx of neutrophils that form a sterile abscess within 2 hours

2001

130. Based on the pathogenesis of failure of passive transfer (FPT), which of the following is considered the absolute critical timeline for a newborn calf to ingest colostrum for maximum IgG absorption?
(A) Between 24 and 48 hours of life
(B) At precisely 72 hours of life
(C) Within the first 6 to 12 hours of life
(D) Any time during the first week of life

2021

131. Which of the following statements provides the best definition of "colostral immunity"?
(A) The passive transfer of maternal antibodies, primarily IgG, ingested by the neonate
(B) The active immunity generated by the neonate's T cells following ingestion of bacteria
(C) The specific systemic suppression of neonatal immune responses caused by milk proteins
(D) The transplacental transfer of memory B cells directly into developing fetal circulation

NODIA

2002

132. What is the defining characteristic of a "hapten" in the context of contact dermatitis or drug-induced hypersensitivity?
(A) A massive, complex protein capable of strongly activating T helper cells directly without prior APC processing
(B) A specific type of autoantibody targeting host DNA structures and causing systemic lupus erythematosus
(C) An innate immune pattern-recognition receptor that binds viral double-stranded RNA in host cytoplasm
(D) A small, non-immunogenic molecule that elicits an immune response only when conjugated to a carrier protein

(A) Neutrophils are present in circulation but lack the critical lysosomal enzymes required for intracellular killing
(B) The primary immunodeficiency prevents the bone marrow from stimulating production of opsonizing antibodies
(C) The neutrophils are hyperactive and directly destroy healthy host barrier tissues, allowing secondary infections
(D) Neutrophils cannot exit the bloodstream to reach infection sites due to defective adhesion molecules

2023

104. Chediak-Higashi syndrome, seen in cattle and certain other species, is primarily categorized by which cellular functional defect?
(A) Complete developmental failure of active mature B-lymphocyte differentiation into antibody-producing plasma cells
(B) Abnormal intracellular granule fusion forming giant lysosomes in phagocytes, impairing chemotaxis and cell killing
(C) Uncontrolled proliferation of functional natural killer cells, leading directly to large granular lymphocytic leukemia
(D) Inability of the host hepatocytes to synthesize classical pathway complement proteins, specifically active C1q

2001

105. Consider the secondary (acquired) immunodeficiency mechanisms:
I. They are inherently genetic and strictly inherited via Mendelian genetics.
II. They can be induced by viral infections targeting cells of the immune system.
III. Severe malnutrition cannot clinically alter immune competence.
IV. Chronic stress and elevated corticosteroids can induce profound secondary immunodeficiency.
V. Feline Immunodeficiency Virus (FIV) is a classic cause of acquired immunodeficiency.
Which of the above statement(s) is/are INCORRECT?
(A) I and III
(B) II, IV and V
(C) I, III and V
(D) II and III

2012

106. By what primary mechanism does the Bovine Viral Diarrhea Virus (BVDV) induce secondary immunosuppression in acutely infected cattle?
(A) By damaging host cell DNA, rapidly transforming normal cells into neoplastic lines
(B) By inducing lymphocyte apoptosis and impairing macrophage and neutrophil functions
(C) By acting as a potent superantigen, triggering overwhelming systemic shock cascades
(D) By binding directly to circulating IgG and IgM, neutralizing plasma antibodies

2004

107. Feline Immunodeficiency Virus (FIV) establishes an acquired immunodeficiency state primarily by infecting and gradually depleting which critical immune cell population?
(A) $CD4^+$ T-helper lymphocytes
(B) Intestinal goblet cells
(C) Epidermal Langerhans cells
(D) Circulating eosinophils

2011

108. Infectious Bursal Disease (IBD) virus in poultry causes severe, prolonged secondary immunodeficiency by destroying precursor cells located in which organ?
(A) Thymus
(B) Bone marrow
(C) Bursa of Fabricius
(D) Spleen

NODIA

2007

109. Which of the following represents an accurate inference regarding the impact of Canine Distemper Virus (CDV) on the canine immune system?
(A) CDV enhances systemic autoimmune pathways, causing fatal hemolytic anemia
(B) CDV targets only nervous tissues, leaving peripheral immune responses fully intact
(C) CDV induces permanent, inherited genetic mutations in offspring of infected dogs
(D) CDV targets lymphoid tissue, causing lymphopenia and secondary bacterial infections

2015

110. In livestock, chronic environmental stress (e.g., overcrowding, extreme temperatures) leads to defective immunocompetence primarily via the sustained release of which physiological compound?
(A) Endogenous histamine
(B) Insulin
(C) Glucocorticoids (e.g., cortisol)

on the detection of which specific type of autoantibody in the serum?
(A) Anti-acetylcholine receptor antibodies
(B) Antinuclear antibodies (ANA)
(C) Anti-desmoglein antibodies
(D) Anti-erythrocyte antibodies

2025
154. What is the fundamental cause-and-effect relationship that results in allergic contact dermatitis to cheap metal collars containing nickel?
(A) Nickel directly lyses the epidermal cells, exposing underlying tissues to immediate destruction by free radicals
(B) Nickel acts as a hapten, binding skin proteins to trigger a Th1-mediated delayed hypersensitivity response
(C) Nickel rapidly binds directly to free IgE in the blood, precipitating as massive immune complexes in capillaries
(D) Nickel physically blocks all local complement regulatory proteins, causing spontaneous autolysis of dermal tissue

2019
155. Consider the following statements concerning the properties of innate immunity:
I. It possesses strict, highly specific memory against unique antigenic epitopes.
II. It responds rapidly using pre-formed receptors (like Toll-like receptors) that recognize broad pathogen-associated molecular patterns (PAMPs).
III. It involves physical barriers, phagocytic cells, and the complement system.
Which of the above statements are correct?
(A) I and II
(B) I and III
(C) II and III
(D) I, II and III

2021
156. Which of the following represents the LEAST relevant finding when investigating a suspected case of primary Severe Combined Immunodeficiency (SCID)?
(A) Profound and persistent systemic lymphopenia in a newborn animal
(B) Severe, recurrent clinical infections despite aggressive therapies
(C) An immediate allergic wheal and flare reaction to flea bites
(D) Severe developmental hypoplasia of primary lymphoid tissue on necropsy

2003
157. An experimental drug perfectly blocks the function of the J-chain. What specific immunological consequence can be reliably inferred?
(A) Complete and permanent inability of activated macrophages to phagocytize extracellular bacteria
(B) Total failure of the classical complement pathway cascade to initiate upon antigen binding
(C) Instantaneous and widespread systemic degranulation of all sensitized tissue mast cells in vivo
(D) Inability of IgM to form pentamers and IgA to form dimers, severely impairing mucosal defense

2007
158. Which of the following is considered an INCORRECT pairing between an innate immune component and its primary functional mechanism?
(A) Lysozyme : Cleaves peptidoglycan in bacterial cell walls
(B) Toll-like receptor 4 (TLR-4) : Recognizes lipopolysaccharide (LPS) of Gram-negative bacteria
(C) C-reactive protein (CRP) : Acts as the primary somatic hypermutation enzyme in memory B cells
(D) Interferon-α/beta : Induces an antiviral state in neighboring uninfected cells

2010
159. The primary biological role of the eosinophil in veterinary immunology is most firmly associated with:
(A) The continuous clearance of small, soluble circulating immune complexes from capillaries
(B) Destruction of multicellular helminths and participation in late-phase allergic reactions
(C) The presentation of processing viral antigens to naive T lymphocytes in regional lymph nodes
(D) The active intracellular synthesis and secretion of high-affinity IgG antibodies in tissue

2005
160. Which sequence accurately orders the classical complement pathway activation events?
(A) C3 spontaneously hydrolyzes → Factor B binds → Factor D cleaves → C3bBb forms

Downloaded from www.pdf.tube

antibody (like IgM) in the context of diagnostic agglutination tests?
(A) Incomplete antibodies lack the Fc region entirely, preventing complement binding and opsonization
(B) They bind target antigens but fail to cross-link them without adding an anti-species globulin
(C) Complete antibodies trigger massive cellular proliferation cascades rather than simple agglutination
(D) Incomplete antibodies bind exclusively to complex carbohydrate antigens and never to proteins

2018
118. When assessing an inflammatory response, which effector cell is LEAST relevant to a pure Type IV delayed-type hypersensitivity reaction?
(A) Macrophages
(B) Epithelioid cells
(C) Basophils
(D) $CD4^+$ T-helper 1 cells

2022
119. A major advantage of colostral immunity over transplacental immunity for a neonate is:
(A) Colostrum relies strictly on active transport of functional memory B cells across the gut lumen
(B) Transplacental pathways only transfer IgM, whereas colostrum selectively delivers protective IgE
(C) Colostral protection is permanent, negating the need for neonates to develop active immunity
(D) Colostrum provides both systemic IgG and localized secretory IgA protecting the neonatal gut

2012
120. Which of the following is a direct, negative consequence of an animal developing a secondary immunodeficiency due to chronic viral infection?
(A) Reduced efficacy of prophylactic vaccines due to impaired immune response capacity
(B) The immediate and complete shedding of all epidermal tissue layers across the body
(C) Spontaneous, severe hyper-calcification of primary lymphoid tissues like the bursa
(D) Complete and permanent resistance to the development of any future autoimmune disease

2009
121. The alternative complement pathway generates a C3 convertase composed of:
(A) C4b and C2a
(B) C3b and Bb
(C) C1q, C1r, and C1s
(D) C5b, C6, C7, C8, and C9

2010
122. Which process defines the physiological concept of "opsonization"?
(A) The direct binding of a foreign antigen to CD4 receptors on helper T lymphocyte populations
(B) Coating a pathogen with IgG or C3b to enhance phagocytosis by macrophages and neutrophils
(C) The intracellular synthesis and release of specific antiviral cytokines by infected cells
(D) The rapid enzymatic degradation of circulating histamine by mucosal histaminase enzymes

NODIA

2008
123. Which of the following statements about Natural Killer (NK) cells is INCORRECT?
(A) They require prior sensitization and antigen-specific TCR binding to kill targets
(B) They are cytolytic cells morphologically classified as large granular lymphocytes
(C) They utilize intracellular perforin and granzymes to trigger apoptosis in targets
(D) They secrete high levels of IFN-gamma, which assists in activating macrophages

2005
124. During a Type I hypersensitivity reaction, the late-phase response (occurring hours after the initial exposure) is predominantly characterized by:
(A) Immediate, explosive release of pre-formed histamine from sensitized mucosal mast cells
(B) Massive deposition of circulating IgG-antigen complexes within local capillary walls
(C) Tissue infiltration by eosinophils and neutrophils, recruited by early-phase mediators
(D) The rapid and organized formation of granulomas by activated epithelioid macrophages

2019
125. Match the immune cells in List I with their primary role or defining marker in List II, and select the correct option.
List I: (Immune Cells)

(D) Thyroid hormone

2022

111. High levels of circulating corticosteroids induce immunosuppression through which of the following cellular effects?

(A) Extensive proliferation of memory B cells and subsequent antibody hypersecretion

(B) Down-regulation of pro-inflammatory cytokines and induction of lymphocyte apoptosis

(C) Up-regulation of MHC class II molecules on the membranes of all dendritic cells

(D) Immediate and irreversible degranulation of all resident tissue mast cell populations

2022

112. Which nutritional factor is INCORRECTLY matched with its role in maintaining immunocompetence in livestock?

(A) Vitamin E/Selenium - Acts as crucial antioxidants to protect phagocyte membranes from self-inflicted oxidative damage during the respiratory burst

(B) Protein-energy malnutrition - Severely reduces lymphocyte proliferation and delays cell-mediated immune responses

(C) Zinc - Essential cofactor for numerous enzymes involved in cell replication and normal thymic function

(D) Copper deficiency - Generally enhances macrophage phagocytosis and increases overall disease resistance

2018

113. Compare the immune consequences of a primary immunodeficiency (such as Bovine Leukocyte Adhesion Deficiency, or BLAD) with a secondary immunodeficiency (such as corticosteroid-induced stress). Which statement correctly distinguishes them?

(A) BLAD is an irreversible genetic defect, while stress is an acquired and reversible state

(B) BLAD targets only the adaptive system, while stress solely impacts innate immune pathways

(C) Both conditions are highly responsive to and completely resolved by standard antimicrobials

(D) BLAD can be prevented by routine viral vaccination, whereas stress requires husbandry changes

2025

114. If a herd of cattle is fed a diet severely deficient in Vitamin E and Selenium, what is the most appropriate predicted outcome regarding their immune function?

(A) Total cessation of immunoglobulin synthesis in lymph nodes, leading to rapid systemic bacterial sepsis

(B) Spontaneous development of severe combined immunodeficiency with complete depletion of T and B cells

(C) Impaired neutrophil bactericidal activity due to oxidative damage, increasing clinical mastitis risk

(D) Immediate and irreversible breakdown of immune tolerance, causing acute systemic lupus erythematosus

2019

115. Consider the following Assertion (A) and **Reason (R):**

Assertion (A): Toxins such as aflatoxin B1 in contaminated feed can cause significant secondary immunodeficiency in poultry and swine.

Reason (R): Aflatoxins impair protein synthesis and suppress cell-mediated immunity, macrophage function, and specific antibody production, increasing susceptibility to infectious diseases.

(A) Both A and R are true and R is the correct explanation of A.

(B) Both A and R are true but R is not the correct explanation of A.

(C) A is true but R is false.

(D) A is false but R is true.

2008

116. Which pair represents an INCORRECT association concerning immunoglobulins and immune system ontogeny?

(A) Epitheliochorial placenta : Prevents pre-natal IgG transfer across maternal-fetal tissues

(B) IgM : Predominant immunoglobulin isotype synthesized during primary immune responses

(C) Fetal B cells : Express surface membrane-bound IgM and IgD during early development

(D) Fetal lymphoid development : Humoral immunity completely matures before innate immunity starts

2017

117. What distinguishes an incomplete antibody (like some anti-erythrocyte IgG) from a complete

Click Here to Buy book on Amazon

2004

133. Which of the following is considered an INCORRECT pair regarding the ontogeny of primary lymphoid organs?
(A) Thymus : Primary site of T cell maturation
(B) Bursa of Fabricius : Primary site of T cell maturation in birds
(C) Bone Marrow : Primary site of B cell maturation in most mammals
(D) Peyer's Patches : Important site of B cell development in ruminants

2025

134. Which phenomenon logically explains why a massive, acute systemic release of histamine during anaphylaxis causes severe, life-threatening hypotension?
(A) Histamine directly stimulates massive, rapid diuresis by the kidneys, depleting blood volume in minutes
(B) Histamine physically binds all available free iron in the blood, completely starving the cardiac muscle
(C) Histamine causes systemic vasodilation and increased vascular permeability, reducing peripheral resistance
(D) Histamine strictly causes complete systemic vasoconstriction, stopping blood flow to vital organs entirely

2001

135. Match the specialized Antigen-Presenting Cell in List I with its characteristic anatomical location in List II, and select the correct option.
List I: (Antigen-Presenting Cell)
P. Langerhans cells
Q. Kupffer cells
R. Microglia
S. Follicular dendritic cells
List II: (Anatomical Location)
1. Liver
2. Epidermis
3. Lymph node follicles
4. Central Nervous System
(A) P-4, Q-1, R-2, S-3
(B) P-2, Q-4, R-1, S-3
(C) P-3, Q-1, R-4, S-2
(D) P-2, Q-1, R-4, S-3

2025

136. Which classification best applies to the immunodeficiency observed in a calf infected in utero with Bovine Viral Diarrhea Virus (BVDV) that remains persistently infected and tolerant to the virus for life?
(A) Pathogen-specific acquired immunotolerance combined with increased risk for secondary infections
(B) Congenital primary immunodeficiency affecting both humoral and cell-mediated lineages from birth
(C) Acute Type III hypersensitivity reaction causing systemic deposition of circulating complexes
(D) Severe generalized genetic neutrophil defect impairing phagocytosis and intracellular killing

2006

137. Which of the following represents an accurate cause-and-effect relationship regarding maternal antibody interference?
(A) Maternal IgG permanently suppresses the neonate's genetic ability to ever form antibodies to that antigen
(B) Maternal IgG binds vaccine antigens, preventing active immunization and causing vaccine failure if given too early
(C) Maternal antibodies physically destroy the neonatal thymus, permanently delaying cell-mediated immune maturity
(D) Maternal IgA directly triggers an immediate Type I hypersensitivity in the neonatal gut, causing severe diarrhea

2001

138. The primary mechanism by which an adjuvant enhances the immune response to a killed vaccine is by:
(A) Serving directly as the primary target antigen recognized by host cytotoxic T lymphocytes
(B) Creating an antigen depot effect and stimulating innate receptors to increase APC activation
(C) Diluting the antigen concentration to prevent immediate Type I anaphylactic reactions in vivo
(D) Completely bypassing T helper cell pathways to induce direct B cell isotype switching in lymph nodes

NODIA

2012

139. Which of the following features is LEAST relevant to the normal physiological function of a mature resting macrophage?
(A) Presentation of endogenous antigens strictly on MHC Class I
(B) Phagocytosis of cellular debris and apoptotic host cells
(C) Production of acute phase cytokines like

IL-1 and TNF-alpha upon activation
(D) Presentation of exogenous antigens on MHC Class II to helper T cells

2009
140. Which specific immune mechanism is most directly responsible for the classic "blue eye" (corneal edema) sometimes seen in dogs recovering from canine adenovirus type 1 (CAV-1) infection or vaccination?
(A) Direct, massive viral replication bursting corneal endothelial cells and disrupting local fluid transport mechanisms
(B) Cross-linking of antigen-specific IgE on sensitized mast cells located throughout the local conjunctival tissues
(C) Deposition of antigen-antibody complexes in the uvea, causing Type III hypersensitivity and complement activation
(D) Direct attack by cytotoxic $CD8^+$ T lymphocytes on lens capsule proteins, causing secondary corneal inflammation

2011
141. Identify the INCORRECT statement regarding the nature of colostrum compared to mature milk.
(A) Colostrum contains a significantly higher concentration of immunoglobulins, particularly IgG in ruminants
(B) Colostrum is rich in maternal lymphocytes, cytokines, and growth factors that aid the neonate
(C) The production of true colostrum ceases shortly after parturition, transitioning to lower-protein mature milk
(D) The immunoglobulin concentration in colostrum continuously increases for the first two weeks of lactation

2023
142. The process of central tolerance in B-lymphocyte development primarily occurs in which anatomical location in most domestic mammals?
(A) The thymic cortex
(B) The red pulp of the spleen
(C) The bone marrow
(D) The mesenteric lymph nodes

2007
143. Which of the following represents a correct pair regarding cellular markers and their specific targets?
(A) CD4 : Binds specifically to conserved regions of host MHC Class I molecules
(B) CD3 : Forms a signaling complex with the T-cell receptor on mature T cells
(C) CD8 : Binds specifically to conserved regions of host MHC Class II molecules
(D) CD21 : Acts as the primary signaling receptor on cytotoxic Natural Killer cells

2016
144. Which process represents a critical elimination mechanism by which the immune system resolves acute inflammation after a pathogen is cleared?
(A) Massive, indefinite up-regulation of IL-2 cytokine production by all stimulated T cell populations
(B) Spontaneous and widespread formation of systemic granulomas to encapsulate completely healthy tissue
(C) Conversion of all antigen-specific plasma cells directly into long-lived tissue mast cell populations
(D) Apoptosis of short-lived effector lymphocytes, contracting the response to leave memory cells

2001
145. Consider the following Assertion (A) and **Reason (R):**
Assertion (A): The administration of fresh frozen plasma is a common and effective treatment for failure of passive transfer in foals older than 24 hours.
Reason (R): Foals older than 24 hours can efficiently absorb immunoglobulins directly across the mature gut epithelium via active pinocytosis.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2005
146. A direct consequence of a defect in the Fas-FasL pathway (such as the lpr mutation in mice) is most likely to be:
(A) Complete inability of plasma cells to synthesize and secrete any functional immunoglobulins
(B) Immediate and total failure of all macrophage and neutrophil innate phagocytosis mechanisms
(C) Failure of peripheral tolerance, causing lymphoproliferation and severe autoimmune disease

Downloaded from www.pdf.tube

(D) Accelerated and heavily enhanced differentiation and production of functional tissue mast cells

2015

147. Which comparison accurately describes the functional difference between IgM and IgG?

(A) IgM mediates efficient complement activation; IgG is the main isotype for systemic opsonization and memory

(B) IgM is a high-affinity monomer in secondary responses; IgG is a low-affinity pentamer in primary responses

(C) IgM acts as the primary molecular mediator of allergic reactions; IgG mediates mucosal defense exclusively

(D) IgM crosses the hemochorial placental barrier easily; IgG is strictly confined to maternal blood vessels

2024

148. Which concept best explains why a primary exposure to an antigen takes 7-14 days to produce a detectable antibody titer, while a secondary exposure produces a robust titer in 2-3 days?

(A) The secondary response utilizes entirely different, pre-formed innate receptors that bypass standard APCs

(B) The pathogen replicates significantly faster during secondary exposure, forcing the host into a rapid response

(C) The bone marrow releases massive stores of pre-formed, non-specific antibodies during any secondary exposure

(D) The presence of antigen-specific memory cells requiring less activation time and undergoing rapid expansion

2013

149. Identify the INCORRECT pair regarding the structural components of an antibody and their specific roles.

(A) Variable (V) regions : Determine antigen-binding specificity

(B) Disulfide bonds : Prevent any flexibility in the hinge region to keep the molecule rigid

(C) Constant (C) regions of heavy chains : Determine the antibody isotype and biological effector function

(D) Hypervariable regions (CDRs) : Form the exact physical contact site with the antigen epitope

2024

150. In a typical Type II hypersensitivity reaction directed against circulating erythrocytes, what is the primary role of the spleen?

(A) It serves as the primary site where allergen-specific IgE cross-links on splenic mast cell populations

(B) It rapidly synthesizes massive amounts of soluble complement regulatory proteins to protect erythrocytes

(C) It acts as the primary site for macrophage-mediated extravascular phagocytosis of opsonized erythrocytes

(D) It actively pumps massive, uncontrolled amounts of vasoactive histamine directly into the venous return

2008

151. Which sequence appropriately tracks the route of a mucosal IgA molecule from synthesis to the intestinal lumen?

(A) Synthesized by enterocytes → diffuses into mucosal blood vessels → binds pathogens → excreted via bile

(B) Synthesized in bone marrow → transported via plasma → direct passive diffusion through tight junctions

(C) Synthesized by intraepithelial T cells → packaged into exosomes → exocytosed directly into gut lumen

(D) Synthesized by plasma cells → binds basolateral poly-Ig receptor → transcytosed and released into lumen

2015

152. Which statement accurately describes a key characteristic of the Major Histocompatibility Complex (MHC) molecules?

(A) MHC Class I presents endogenous peptides to $CD8^+$ T cells; MHC Class II presents exogenous peptides to $CD4^+$ T cells

(B) They are highly conserved proteins, displaying virtually identical genetic sequences across all individuals

(C) Both Class I and Class II molecules are strictly and exclusively expressed on professional antigen-presenting cells

(D) They bind directly and with high affinity to intact, un-processed globular protein antigens floating in plasma

2010

153. A positive diagnosis of Systemic Lupus Erythematosus (SLE) in a dog strongly relies

NODIA

Desmoglein 3 protein
(C) Systemic Lupus Erythematosus : Intracellular double-stranded DNA
(D) Myasthenia gravis : Post-synaptic acetylcholine membrane receptors

2004
97. If an animal is diagnosed with acquired myasthenia gravis, which of the following clinical observations directly correlates with the immunopathological cause?
(A) Massive accumulation of circulating antigen-antibody complexes depositing in the filtration barrier of renal glomeruli
(B) Severe epidermal blistering and cleft formation due to the autoimmune-mediated destruction of hemidesmosomes
(C) Reversible muscle weakness after exercise due to autoantibody blockade of acetylcholine receptors
(D) Rapid, complement-mediated intravascular hemolysis following sudden host exposure to freezing cold environmental temperatures

2017
98. Severe Combined Immunodeficiency (SCID) in Arabian foals is classified as a primary congenital immunodeficiency because it is caused by:
(A) A severe maternal nutritional deficiency of zinc and selenium during critical late gestation
(B) An autosomal recessive mutation in the DNA-PKcs gene, disrupting V(D)J recombination
(C) An overwhelming, acute in utero pathogen infection by the cytopathic Equine Herpesvirus-1
(D) Active transplacental transfer of maternal IgG autoantibodies that destroy the developing fetal thymus

2009
99. Which of the following is the most obvious clinicopathological consequence observed in a foal born with Arabian SCID?
(A) Massive lymphadenopathy and splenomegaly due to completely unregulated and malignant lymphocyte proliferation
(B) Severe autoimmune destruction of the foal's own circulating erythrocytes, rapidly leading to acute fatal anemia
(C) Spontaneous systemic bleeding diatheses and hemorrhage due to a complete lack of coagulation factor VIII
(D) Absence of B and T lymphocytes, leading to fatal opportunistic infections as colostral antibodies wane

2001
100. Choose the sequence of diagnostic findings that logically confirms a diagnosis of SCID in an Arabian foal.
(A) Profound lymphopenia → Hypogammaglobulinemia post-weaning → Confirmation of DNA-PKcs mutation via PCR
(B) Elevated total white cell count → Abundant presence of LE cells in fluid → Positive direct Coombs test
(C) Positive tuberculin skin test response → High levels of antigen-specific IgE → Marked hyperglobulinemia
(D) Low systemic hematocrit → High circulating reticulocyte count → Abundant presence of spherocytes on blood smear

2013
101. Selective IgA deficiency, frequently observed in certain dog breeds like the German Shepherd, primarily results in an increased clinical susceptibility to which type of conditions?
(A) Severe, rapidly progressive and fatal systemic opportunistic bacteremia and sepsis
(B) Widespread, painful cutaneous blistering and deep chronic tissue granulomas
(C) Recurrent mucosal infections, including chronic enteritis and respiratory disease
(D) Immediate, acute systemic anaphylactic shock reactions to routine core vaccinations

2008
102. Which statement INCORRECTLY characterizes Bovine Leukocyte Adhesion Deficiency (BLAD)?
(A) It causes an absolute neutropenia (lack of neutrophils) in the circulating peripheral blood
(B) It is an autosomal recessive genetic disorder affecting Holstein cattle
(C) It involves a point mutation in the gene encoding the CD18 integrin subunit (beta-2 integrin)
(D) It results in an inability of neutrophils to undergo tight adhesion and diapedesis across the vascular endothelium

2023
103. If a Holstein calf possesses the BLAD mutation, what is the cause-and-effect relationship regarding its chronic, severe infections despite having high numbers of circulating white blood cells?

Click Here to Buy book on Flipkart

→ C1q activates
(B) C5 binds antigen → recruits C6-C9 → forms MAC → C3 convertase activates → C1q stabilizes
(C) C4b2a forms → C1q binds → MAC assembles → C3b opsonization → C5a releases
(D) C1q binds Fc → C1r/s activated → C4/C2 cleaved → C3 convertase formed → C3 cleaved → C5 convertase formed

2006

161. A specific defect in the process of somatic hypermutation within germinal centers will directly prevent which of the following outcomes?
(A) The occurrence of affinity maturation and subsequent production of high-affinity IgG antibodies
(B) The initial generation and genetic recombination of a vast repertoire of naïve B cell receptors
(C) The physical ability of a secreted antibody to bind directly to the complement C1q initiator
(D) The basic capability of mature B cells to undergo class switch recombination from IgM to IgE

2007

162. Which of the following defines an INCORRECT concept regarding maternal immunity and vaccination?
(A) Maternal antibodies provide vital early protection but have a finite half-life and will eventually wane
(B) The exact time maternal antibodies fall below a protective level varies significantly among individuals within a litter
(C) A single vaccination at 4 weeks of age guarantees permanent immunity for all puppies, regardless of maternal antibody levels
(D) The "window of susceptibility" occurs when maternal antibodies are too low to protect against wild infection, but still high enough to block successful vaccination

2013

163. The Arthus reaction is primarily driven by:
(A) Subcutaneous immune complex formation with circulating IgG, activating complement and neutrophils
(B) Massive and rapid release of vasoactive histamine locally due to antigen-specific IgE cross-linking
(C) Widespread systemic vasodilation triggered by soluble immune complexes depositing in central vessels
(D) Local activation of memory Th1 cells and extensive macrophage infiltration occurring 72 hours later

2004

164. In the Gell and Coombs classification, autoimmune hemolytic anemia (AIHA) and neonatal isoerythrolysis share which underlying mechanism?
(A) Type I hypersensitivity
(B) Type III hypersensitivity
(C) Type IV hypersensitivity
(D) Type II hypersensitivity

2001

165. Consider the following statements regarding autoimmune diseases:
I. Autoimmunity represents a breakdown of self-tolerance.
II. SLE is categorized as an organ-specific autoimmune disease strictly affecting the thyroid.
III. Autoantibodies in Myasthenia Gravis target the neuromuscular junction.
IV. Pemphigus foliaceus involves Type II hypersensitivity mechanisms targeting the epidermis.
Which of the above statements is/are INCORRECT?
(A) I and II
(B) II only
(C) II and III
(D) III and IV

2022

166. Which process is an essential elimination mechanism allowing cytotoxic $CD8^+$ T cells to effectively kill virally infected cells without destroying themselves in the process?
(A) The complete shedding of their own cellular membrane before initiating cytolytic attacks on target cells
(B) The rapid, systemic secretion of highly caustic acids directly into the general bloodstream of the host
(C) The directional release of perforin and granzymes into the immunological synapse with the target cell
(D) The internalization of the virus into the T cell itself, sacrificing the T cell instead of the host tissue

Downloaded from www.pdf.tube

signals to naive B lymphocytes. This interaction drives the clonal expansion and terminal differentiation of these B cells into antibody secreting plasma cells. These specialized effector cells produce large quantities of soluble immunoglobulins designed to target and eliminate foreign antigens throughout the host organism.

10. (B) III, I, IV, II
B lymphocyte development follows a strict chronological sequence of maturation. The process initiates with pro B cells, which progress into pre B cells characterized by pre B cell receptor expression. These cells then develop into immature B cells in the bone marrow, expressing membrane IgM, and finally mature into naive B cells co expressing surface IgM and IgD.

11. (B) By detecting down-regulation of host MHC class I molecules
Natural Killer cells monitor healthy cells using inhibitory receptors that recognize major histocompatibility complex class I molecules. Many viruses down regulate these surface molecules to escape cytotoxic T lymphocytes. This absence of major histocompatibility complex class I relieves the inhibitory signal on Natural Killer cells, triggering the activation of their cytolytic machinery to destroy the infected cell.

12. (B) cDCs present antigen in lymph nodes, while macrophages present to T cells in peripheral tissues
Conventional dendritic cells are specialized to capture antigens in peripheral tissues and migrate to draining lymph nodes to activate naive T lymphocytes. In contrast, tissue macrophages typically remain resident within peripheral tissues, presenting antigens locally to effector and memory T lymphocytes to coordinate immediate, localized immune responses at the site of pathogen entry or inflammation.

13. (A) Direct somatic hypermutation of its T-cell receptor genes
Effector cytotoxic T lymphocytes utilize stable T cell receptors that do not undergo somatic hypermutation after leaving the thymus. Somatic hypermutation is a process unique to B lymphocytes within germinal centers, designed to increase antibody affinity. T lymphocytes rely instead on clonal selection of pre existing receptors and unleash effector mechanisms like perforin and granzyme secretion upon activation.

14. (C) The T-lymphocytes will enter a state of anergy due to the lack of costimulation
Naive T lymphocytes require two signals for activation: antigen recognition via the T cell receptor and costimulation through CD28 binding to B7 ligands on antigen presenting cells. Blocking CD28 prevents this critical second signal. Consequently, instead of activating, the T lymphocytes become functionally unresponsive or anergised, preventing an effective adaptive immune response.

15. (B) II and III
Helper T cells belonging to the Th1 lineage specialize in supporting cell mediated immunity by secreting interferon gamma and interleukin 2. Regulatory T cells maintain peripheral immunological tolerance and control inflammatory responses, characteristically expressing CD4, CD25, and the transcription factor FoxP3. In contrast, $CD4^+$ T cells recognize antigens presented by major histocompatibility complex class II molecules.

16. (D) It presents exogenous viral antigens via MHC class I to activate naive $CD8^+$ T cells without direct cell infection
Cross presentation allows professional dendritic cells to acquire exogenous viral antigens from extracellular spaces or dying cells and process them into the major histocompatibility complex class I pathway. This mechanism enables the activation of naive $CD8^+$ cytotoxic T lymphocytes to combat intracellular viral infections without requiring the dendritic cell itself to be directly infected by the pathogen.

17. (C) Bursa of Fabricius
The Bursa of Fabricius is a unique, specialized primary lymphoid organ found exclusively in avian species. Located near the cloaca, it provides the essential microenvironment required for the proliferation, gene conversion, and maturation of avian B lymphocytes. Surgical or viral destruction of this organ impairs the development of humoral immunity and antibody production.

18. (B) Phagocytosis followed by enzymatic degradation within the phagolysosome
Macrophages recognize opsonized pathogens through surface Fc receptors or complement

NODIA

2012

174. Which process accurately describes the role of the poly-Ig receptor in mucosal immunity?

(A) It binds monomeric IgG in the gut lumen, transporting it actively across enterocytes into the bloodstream

(B) It acts as the primary signaling receptor on follicular B cells to initiate somatic hypermutation events

(C) It binds dimeric IgA in the lamina propria, transporting it across epithelial cells to mucosal surfaces

(D) It actively degrades free IgE in the respiratory tract, preventing the development of chronic asthma

2005

175. Consider the following statements concerning the properties of maternal immunity in dogs:

I. Puppies receive roughly 5-10% of their maternal immunity via the endotheliochorial placenta.

II. The majority of maternal antibodies are absorbed via colostrum during the first 24 hours of life.

III. High levels of maternal antibodies can neutralize vaccine antigens, causing vaccine failure in young puppies.

IV. Gut closure prevents the continued absorption of intact maternal IgG after approximately one day.

Which of the above statements are correct?

(A) I, II, III and IV

(B) II, III and IV

(C) I, II and IV

(D) I, III and IV

2003

176. In an experimental model, knocking out the gene for the TAP (Transporter associated with Antigen Processing) protein would result in which immediate functional defect?

(A) Failure to load exogenous peptides onto MHC Class II molecules, blocking $CD4^+$ T helper cell activation pathways

(B) Total failure of plasma cells to secrete intact, functional immunoglobulins into the surrounding lymph fluid

(C) Immediate, spontaneous destruction and cellular apoptosis of the thymic cortex during early T cell selection

(D) Failure to transport endogenous peptides into the endoplasmic reticulum, preventing MHC Class I expression

2007

177. Which statement provides the most accurate definition of an "autoantigen"?

(A) An intrinsic host molecule that becomes the target of an aberrant autoimmune response

(B) A completely foreign viral protein that mimics a host tissue structure, triggering immunity

(C) A specific environmental chemical that acts as a hapten, binding carrier proteins in skin

(D) An artificially synthesized protein compound designed and used exclusively in modern vaccines

2003

178. What is the fundamental consequence of a mutation in the FOXP3 gene (such as in scurfy mice or IPEX syndrome)?

(A) Failure of the classical complement pathway, preventing antibody-mediated pathogen lysis

(B) Total absence of all functional B lymphocytes in the bone marrow and peripheral tissues

(C) Absence of functional regulatory T cells, causing uninhibited multi-organ autoimmunity

(D) Inability to form memory T cells, causing repeated clinical infections with the same pathogen

2002

179. Which of the following defines an INCORRECT concept regarding the complement Membrane Attack Complex (MAC)?

(A) The MAC relies primarily on the structural scaffolding of circulating IgE to hold it against the cell membrane

(B) The MAC requires the sequential assembly of C5b, C6, C7, C8, and multiple C9 molecules

(C) The MAC forms a stable transmembrane pore that disrupts the osmotic integrity of the target cell

(D) The MAC is generally more effective at lysing Gram-negative bacteria than Gram-positive bacteria due to the exposed outer lipid membrane

2002

180. The classical phenomenon of "serum sickness" following administration of large amounts of foreign antitoxin is characterized by which of the following temporal patterns?

(A) Symptoms strictly occur within 5 minutes of administration and resolve entirely within a single hour

Click Here to Buy book on Amazon

2014

167. Which of the following presents a correct pair regarding cytokines and their primary function?
(A) Interleukin-4 (IL-4) : Promotes Th1 differentiation and classical macrophage activation
(B) Interferon-gamma (IFN-γ) : Promotes B cell isotype switching directly to IgE
(C) Transforming Growth Factor-beta (TGF-β) : Acts as the most potent endogenous pyrogen (fever inducer)
(D) Interleukin-10 (IL-10) : Exerts broadly immunosuppressive and anti-inflammatory effects

2004

168. The primary immunological justification for utilizing a core, multivalent vaccination strategy in young domestic animals (e.g., puppies and kittens) is to:
(A) Induce long-lived protective memory responses against severe pathogens as maternal antibodies decline
(B) Provide a continuous, lifelong stream of artificial passive immunity without active pathogen exposure
(C) Immediately cure and completely clear any pre-existing congenital viral infections they may harbor
(D) Permanently stimulate the innate immune system so the animal never relies on adaptive lymphoid cells

2006

169. What is the fundamental functional difference between primary and secondary lymphoid organs?
(A) Primary organs strictly filter circulating blood, whereas secondary organs strictly filter tissue lymph
(B) Primary organs generate and mature lymphocytes, while secondary organs are where they undergo activation
(C) Primary organs rely exclusively on B cell development, whereas secondary organs rely entirely on T cells
(D) Primary organs are functional only in the fetus, whereas secondary organs develop only in adult animals

2008

170. Which mechanism explains the cause-and-effect relationship of profound immunosuppression in advanced cases of Feline Immunodeficiency Virus (FIV)?
(A) The virus systematically infects and destroys bone marrow erythrocytes, causing fatal anemia and systemic weakness
(B) The virus depletes $CD4^+$ helper T cells, crippling both humoral and cell-mediated adaptive immune responses
(C) The virus massively overstimulates B cells, causing them to exhaust their entire antibody reserves within weeks
(D) The virus directly binds and neutralizes circulating complement proteins, completely blocking inflammation

2002

171. A 6-month-old calf presents with severe, recurrent bacterial pneumonias and extreme neutrophilia (elevated neutrophils in the blood), yet tissue biopsies show no neutrophil infiltration at the sites of infection. This clinical picture is most strongly indicative of:
(A) Bovine Leukocyte Adhesion Deficiency (BLAD)
(B) Severe Combined Immunodeficiency (SCID)
(C) Neonatal isoerythrolysis
(D) Chediak-Higashi Syndrome

NODIA

2004

172. Which of the following is considered an INCORRECT pair concerning the effector mechanisms of hypersensitivities?
(A) Type I : IgE and mast cells
(B) Type II : Cytotoxic IgG/IgM and complement
(C) Type IV : Free-floating soluble histamine directly attacking tissue
(D) Type III : Soluble immune complexes and neutrophil-mediated damage

2018

173. What is the most appropriate description of "epitope spreading" during the progression of an autoimmune disease?
(A) The physical metastasis and cellular migration of active inflammatory autoimmune lesions from the skin to the pulmonary system
(B) The rapid, highly coordinated therapeutic vaccination of an animal against multiple unrelated pathogens simultaneously
(C) The passive vertical transfer of maternal autoimmune antibodies from a gestating dam to her developing fetus across the placenta
(D) The immune response expands from targeting a single self-epitope to attacking additional self-epitopes as tissue damage progresses

circulating neutrophils in peripheral blood throughout the animal's lifetime

2025

187. Bovine Neonatal Pancytopenia (BNP) is an alloimmune disease of calves characterized by systemic hemorrhages and severe bone marrow depletion. What is the primary immunopathogenesis of this condition?

(A) Ingestion of colostrum containing maternal alloantibodies, induced by a specific BVDV vaccine, that target and destroy neonatal leukocyte and megakaryocyte antigens

(B) In utero transmission of a cytopathic virus that selectively destroys the hematopoietic stem cells in the fetal liver

(C) Severe dietary deficiency of iron and copper in the gestating dam, leading to a complete arrest of fetal hematopoiesis

(D) Type I hypersensitivity reaction against bovine serum albumin present in artificial milk replacers

2023

188. Identify the INCORRECT statement concerning Type IV hypersensitivity:

(A) It is often referred to as delayed-type hypersensitivity (DTH)

(B) It involves the activation of memory Th1 cells and the subsequent massive recruitment of activated macrophages

(C) It heavily relies on the massive deposition of circulating antigen-antibody complexes into the tissue capillary beds

(D) It is the primary immune mechanism driving chronic granuloma formation against persistent intracellular pathogens like Mycobacteria

2018

189. A newborn foal is diagnosed with neonatal isoerythrolysis. Which of the following represents the correct sequence of events that led to this condition?

(A) Foal ingests foreign environmental antigens in colostrum → severe Type I systemic shock develops immediately → histamine-mediated RBC lysis occurs

(B) Sire passes defective RBC genes to foal → foal's erythrocytes spontaneously lyse upon first breathing oxygen → severe hemolytic anemia develops

(C) Dam transfers anti-foal RBC specific cytotoxic T-lymphocytes across the placenta → T-cells mature in fetus and destroy bone marrow post-partum

(D) Foal inherits sire's RBC antigen → maternal IgG is produced against it and transferred via colostrum → causes complement hemolysis of foal's RBCs

2024

190. Consider the following progressive stages of an active systemic autoimmune disease, such as Systemic Lupus Erythematosus (SLE):

I. Deposition of immune complexes in filtering capillary beds (glomeruli, synovium).

II. Breakdown of central and/or peripheral immunological tolerance.

III. Activation of the classical complement cascade and neutrophil infiltration.

IV. Production of high-affinity autoantibodies (e.g., antinuclear antibodies).

Which of the following represents the correct sequence of these stages?

(A) II, IV, I, III

(B) IV, II, III, I

(C) I, III, II, IV

(D) II, I, IV, III

2006

191. Which pair accurately matches a primary mediator with its immediate physiological effect during an anaphylactic reaction?

(A) Major Basic Protein : Immediate suppression of T-helper cell function

(B) Histamine : Profound bronchoconstriction and massive vasodilation

(C) Interleukin-2 : Rapid destruction of all host erythrocytes

(D) IgG : Direct, immediate lysis of the inciting allergen

2018

192. Which of the following defines an INCORRECT concept regarding the function of professional antigen-presenting cells (APCs)?

(A) Dendritic cells are considered the most potent APCs for activating naive T cells

(B) Macrophages serve as APCs but typically act upon already-activated effector T cells in peripheral tissues

(C) B cells can act as specialized APCs by capturing specific antigens via their surface immunoglobulins and presenting them to helper T cells

(D) Neutrophils are strictly the most highly efficient and exclusive APCs found within the germinal centers of lymph nodes

Downloaded from www.pdf.tube

(B) Symptoms only appear many years later when host memory T cells finally reactivate spontaneously in vivo
(C) Symptoms require lifelong, continuous daily administration of the foreign protein to ever appear clinically
(D) Symptoms arise 7-14 days later as host IgG forms soluble complexes with remaining foreign proteins

2015

181. Compare the primary functional roles of IgG and IgE in the domestic animal immune system. Which statement is accurate?
(A) IgG is strictly limited to defending mucosal surfaces in the gut, whereas IgE is solely for viral neutralization
(B) IgG dominates systemic opsonization and memory, while IgE is bound to mast cells for parasite and allergic defense
(C) IgG causes massive histamine release during acute shock, whereas IgE directs strict cell-mediated cytotoxicity
(D) Both immunoglobulin isotypes exist primarily as large, complex pentamers in the systemic blood circulation of hosts

2005

182. A severe deficiency in dietary zinc can cause significant secondary immunodeficiency primarily due to its role in:
(A) Maintaining the structural integrity and stable pore-forming capability of the complement MAC in membranes
(B) Acting as an essential structural component of the joining (J) chain in secretory dimeric IgA antibodies
(C) Serving as an essential cofactor for enzymes required for lymphocyte proliferation and thymic function
(D) Directly neutralizing reactive oxygen species and free radicals within the active phagolysosome of neutrophils

2007

183. Which of the following is considered the LEAST relevant clinical finding when suspecting an animal has a primary (congenital) immunodeficiency?
(A) The animal exhibits recurrent, severe infections with opportunistic pathogens that fail to respond normally to standard treatments
(B) The condition typically manifests shortly after maternal colostral antibodies wane
(C) Multiple littermates may exhibit identical severe susceptibility to infections
(D) The animal develops signs of disease only in extreme old age after a lifetime of good health

2012

184. The phenomenon of "molecular mimicry" as a trigger for autoimmunity relies on the fact that:
(A) Structural similarity between a pathogen epitope and a host antigen causes lymphocytes to cross-react
(B) The host tissues purposefully synthesize proteins that mimic viral antigens to trick local immune responses
(C) The host immune system actively mutates its own T-cell receptors during infections to match any somatic protein
(D) Regulatory T cells adopt the exact physical morphology and surface receptor layout of invading bacterial species

2024

185. Consider the following Assertion (A) and **Reason (R):**
Assertion (A): In neonatal foals, the measurement of total serum protein via refractometry is a reliable and frequently used screening tool for assessing the success of passive transfer of colostral immunity.
Reason (R): Immunoglobulins, predominantly IgG absorbed from colostrum, constitute a major fraction of the globulin portion of blood proteins; thus, an adequately nursed foal will show a measurable increase in total serum protein compared to a pre-suckle baseline.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

NODIA

2004

186. Which feature is highly characteristic of Chediak-Higashi syndrome in affected animals?
(A) Absolute inability of mature B lymphocytes to undergo heavy chain class switching to synthesize secretory IgM
(B) Presence of giant fused lysosomal granules in neutrophils and melanocytes, impairing killing and causing albinism
(C) Highly accelerated and highly efficient killing of all intracellular bacterial species within tissue macrophages
(D) Total and persistent absence of any

actively destroys all fetal antibodies to prevent embryonic autoimmunity

2021

199. When considering the role of cytokines in shaping the immune response, which elimination mechanism ensures that a Th1 response suppresses a competing Th2 response?

(A) Th1 cells strictly secrete high concentrations of IL-4, which induces rapid apoptosis of Th2 lymphocytes

(B) Th1 cells physically target, bind, and actively phagocytize competing Th2 cell populations in lymph nodes

(C) Th1 cells rapidly convert directly into mature tissue mast cells to flood the local system with histamine

(D) Th1 cells secrete IFN-gamma, promoting classical macrophage activation and inhibiting Th2 proliferation

2014

200. Identify the INCORRECT statement regarding the basic structure and function of the major immunoglobulin isotypes:

(A) IgD is the primary structural component of the classical complement Membrane Attack Complex

(B) IgA is primarily found as a dimer in mucosal secretions and acts heavily via virus neutralization and preventing pathogen adherence

(C) IgE is present in extremely low concentrations in normal serum but binds with very high affinity to receptors on mast cells

(D) IgM is heavily involved in the primary immune response and acts as an excellent agglutinating and complement-activating antibody

2021

201. Which sequence appropriately tracks the cellular events in a classical cell-mediated Type IV delayed hypersensitivity reaction (e.g., tuberculin test)?

(A) Exposure → binding by allergen-specific IgE → massive histamine release → local wheal and flare at 15 minutes

(B) Re-exposure → Th1 recognition → IFN-gamma secretion → macrophage recruitment/activation → induration at 48-72 hours

(C) Exposure → binding by circulating systemic IgG → complement cascade activation → local neutrophil necrosis within 4 hours

(D) Exposure → binding by cytotoxic T cells → immediate systemic anaphylactic shock and vascular collapse within 1 hour

2003

202. The immunological consequence of a lack of a thymus (e.g., in nude mice or DiGeorge syndrome) is most appropriately described as:

(A) Complete, absolute lack of functional B cells and immunoglobulins, but leaving T cell maturation pathways fully intact

(B) Profound immunodeficiency with absent mature T cells, preventing cell-mediated and T-dependent antibody responses

(C) Massive hypertrophic overgrowth of the avian bursa of Fabricius to physiologically compensate for the missing thymus

(D) A highly specific developmental inability to fight off large parasitic worms, with normal responses to all viral agents

2002

203. Which of the following is an INCORRECT pair concerning the complement system pathways?

(A) Terminal Pathway : Initiated strictly by the direct binding of free histamine to C9

(B) Classical Pathway : Initiated primarily by antigen-antibody complexes (IgG or IgM) binding to C1q

(C) Alternative Pathway : Initiated by spontaneous hydrolysis of C3 and stabilized by pathogenic surfaces

(D) Lectin Pathway : Initiated by mannose-binding lectin attaching to pathogen surface carbohydrates

2022

204. By what physiological mechanism does the phenomenon of "gut closure" fundamentally alter the immunological status of a newborn ruminant?

(A) It permanently prevents the developing neonate from ever actively absorbing any further essential vitamins

(B) It actively triggers the rapid, systemic enzymatic breakdown of all previously absorbed colostral antibodies

(C) Enterocytes capable of pinocytosing immunoglobulins are replaced by mature cells, ending passive transfer

(D) It actively initiates the neonate's own massive and uncoordinated production of systemic IgE immunoglobulins

Click Here to Buy book on Flipkart

2010

193. The primary pathophysiological effect of prolonged stress (elevated endogenous cortisol) on the livestock immune system is:

(A) Severe cytokine down-regulation, lymphocyte suppression, and impaired cellular immunity, increasing susceptibility to infection

(B) Massive, spontaneous over-production of all serum immunoglobulin isotypes, directly leading to systemic hyperviscosity syndrome

(C) The permanent structural destruction of primary lymphoid organs, fully rendering the animal into a severe SCID-like phenotype

(D) An absolute, irreversible block of all innate macrophage phagocytosis without exerting any effect on adaptive lymphoid responses

2012

194. What is the most appropriate description of a "superantigen" and its effect on the immune system?

(A) A tiny mutagenic chemical compound that binds directly to host DNA structures, rapidly mutating genetic material

(B) A strictly highly specific foreign antigen that safely induces perfect, lifelong memory after a single exposure

(C) A protein non-specifically cross-linking MHC Class II to the TCR, causing massive T cell activation and a cytokine storm

(D) An artificially engineered monoclonal antibody that binds to and successfully neutralizes every known host virus

2005

195. Match the veterinary autoimmune disease in List I with its corresponding primary hypersensitivity mechanism in List II, and select the correct option.

List I: (Veterinary Autoimmune Disease)

P. Autoimmune Hemolytic Anemia

Q. Systemic Lupus Erythematosus (vascular lesions)

R. Pemphigus foliaceus

S. Immune-mediated polyarthritis

List II: (Primary Hypersensitivity Mechanism)

1. Type III Hypersensitivity
2. Type II Hypersensitivity

(A) P-2, Q-1, R-2, S-1

(B) P-1, Q-2, R-1, S-2

(C) P-2, Q-2, R-1, S-1

(D) P-1, Q-1, R-2, S-2

NODIA

2009

196. Which sequence most accurately reflects the process of somatic hypermutation and affinity maturation in a responding B cell?

(A) B cell encounters antigen → switches isotype to IgE → mutates heavy chain constant region → induces permanent peripheral tolerance

(B) T cell binds antigen → secretes perforin → responding B cell mutates T cell receptor → phenotypic conversion to a macrophage

(C) B cell replicates in bone marrow → secretes non-specific antibody → highest affinity antibodies selected directly by red blood cells

(D) Antigen binds B cell → germinal center point mutations → high-affinity selection by follicular dendritic cells → differentiation

2023

197. Which of the following statements represents an INCORRECT cause-and-effect relationship regarding maternal immunity?

(A) If a pregnant mare is aggressively vaccinated against tetanus 4 weeks prior to foaling, the resulting colostrum will likely contain very high levels of specific anti-tetanus IgG

(B) If a calf fails to nurse within 24 hours of birth, the inevitable lack of absorbed maternal IgG will strictly cause it to instantly develop a permanent genetic defect in its own B cells

(C) If a puppy nurses adequately from an immune bitch, its high level of circulating maternal antibodies can actively neutralize early doses of a modified-live viral vaccine

(D) If a dam possesses alloantibodies against the sire's blood type, her normal colostrum can become highly toxic to the foal's erythrocytes upon ingestion

2005

198. The most significant immunological difference between an endotheliochorial placenta (dogs/cats) and an epitheliochorial placenta (horses/pigs) regarding passive immunity is:

(A) The epitheliochorial placenta transfers only pure IgE to the fetus, whereas the endotheliochorial transfers only pure IgM

(B) The epitheliochorial placenta transfers 100% of maternal immune cells, completely replacing developing fetal bone marrow

(C) The endotheliochorial placenta allows 5-10% pre-partum IgG transfer, whereas the epitheliochorial blocks all transfer

(D) The endotheliochorial placenta strictly and

ANSWERS

1. (B) Two identical heavy chains and two identical light chains linked by disulfide bonds
Immunoglobulins consist of four polypeptide chains, including two identical heavy chains and two identical light chains. These chains are covalently linked together by interchain disulfide bonds to form a stable monomeric structure. This specific configuration provides the structural framework necessary for precise antigen recognition and subsequent biological effector functions of the antibody molecule.

2. (D) It provides segmental flexibility, allowing the two Fab arms to bind to separate antigenic determinants
The hinge region of certain immunoglobulin classes offers significant segmental flexibility to the antibody molecule. This flexibility allows the two antigen binding fragments to move independently, facilitating binding to epitopes that are spaced at varying distances on a pathogen surface. Consequently, this enhances the efficiency of antigen docking and subsequent immune clearance mechanisms.

3. (A) It requires interaction with the secretory component to function in intravascular spaces
Pentameric immunoglobulin M functions primarily within the vascular system as a highly effective activator of the complement cascade. It does not require a secretory component to operate inside blood vessels, as this specialized polypeptide is instead utilized for transporting polymeric immunoglobulins across mucosal epithelial cells into external secretions. Thus, intravascular activities remain completely independent of secretory components.

4. (C) IgA : Primary secretory antibody protecting mucosal surfaces against enteric pathogens
Secretory immunoglobulin A represents the predominant antibody class found in mucosal secretions, serving as a critical first line of defense at mucosal barriers. It prevents the attachment and colonization of viral and bacterial pathogens in the gastrointestinal, respiratory, and urogenital tracts. This protective mechanism is vital for maintaining mucosal integrity in domestic animals.

5. (A) Secretory component : Drives somatic hypermutation
The secretory component is a glycoprotein that facilitates the transport of polymeric immunoglobulins across epithelial cells and protects them from proteolytic degradation in mucosal environments. It plays no role in driving somatic hypermutation, which is an enzyme driven process occurring inside germinal centers of secondary lymphoid organs to increase antibody affinity for specific antigens.

6. (D) Binding to specific epitopes on pathogenic microorganisms
The crystallizable fragment region of an antibody molecule is responsible for mediating effector functions such as complement activation, opsonization, and cellular cytotoxicity. It does not engage in binding to specific epitopes on pathogenic microorganisms. That specific role is exclusively reserved for the antigen binding fragment regions, which contain the highly variable complementarity determining regions.

7. (B) IgM and IgD
Mature, naive B lymphocytes undergo alternative RNA splicing of a single primary transcript to co express both immunoglobulin M and immunoglobulin D on their cell surface. These surface bound immunoglobulins share identical antigen specificity and function as the primary antigen receptors. Upon antigen encounter and subsequent activation, these cells undergo class switching to produce other isotypes.

8. (C) Dimeric IgA
Dimeric immunoglobulin A is the predominant antibody class secreted across mucosal surfaces, such as the respiratory tract. Its polymeric structure, containing four antigen binding sites, provides high functional avidity for binding and neutralizing viral pathogens before they can adhere to epithelial cells. This localized defense mechanism is crucial for preventing respiratory infections in calves.

9. (D) Antibody-secreting plasma cells
Activated helper T lymphocytes release specific cytokines and provide essential costimulatory

NODIA

2013

205. Match the immune component in List I with its corresponding primary characteristic in List II, and select the correct option.

List I: (Immune Component)

P. Antigen
Q. Antibody
R. Adjuvant
S. Hapten

List II: (Primary Characteristic)

1. Enhances the immunogenicity of a vaccine without being the specific target
2. Small molecule requiring a carrier protein to elicit an immune response
3. Molecule specifically recognized by the adaptive immune system
4. Soluble protein complex secreted by plasma cells

(A) P-4, Q-3, R-2, S-1
(B) P-3, Q-1, R-4, S-2
(C) P-1, Q-4, R-3, S-2
(D) P-3, Q-4, R-1, S-2

2009

206. Which of the following concepts explains why a localized, excessive deposition of immune complexes (the Arthus reaction) leads to severe local tissue necrosis?

(A) The complexes physically suffocate local cells by completely and permanently blocking all microvascular oxygen exchange
(B) The complexes behave as directly toxic, heavy chemical compounds that poison cellular organelles and trigger necrotic lysis
(C) Complexes activate complement, recruiting neutrophils via C5a that release destructive lysosomal enzymes and oxygen radicals
(D) The complexes trigger rapid, massive localized histamine release from resident mast cells that directly digests collagen fibers

2005

207. A primary difference between early-phase and late-phase Type I hypersensitivity reactions is that:

(A) The early phase relies strictly on Th1 inflammatory cytokine release, whereas the late phase relies entirely on Th2 helper cells
(B) Early phase is driven by pre-formed histamine causing vasodilation, while late phase involves lipid mediators and recruited cells
(C) The early phase requires massive classical complement activation, whereas the late phase requires absolutely no humoral proteins
(D) The early phase strictly causes localized dermal eczema, whereas the late phase always and inevitably causes fatal systemic shock

2002

208. Identify the INCORRECT statement regarding Central Tolerance.

(A) It occurs in primary lymphoid organs (thymus for T cells, bone marrow for B cells)
(B) Developing lymphocytes that react with high affinity to presented self-antigens are eliminated primarily via apoptosis (negative selection)
(C) Some self-reactive $CD4^+$ T cells may be diverted to become natural regulatory T cells (Tregs) rather than being deleted
(D) It strictly eliminates perfectly functioning, highly reactive pathogen-specific lymphocytes to save energy

2022

209. What is the fundamental mechanism underlying the pathogenesis of Myasthenia Gravis?

(A) Autoantibodies block and degrade acetylcholine receptors at the neuromuscular junction, causing skeletal muscle weakness
(B) Spontaneous, rapid degeneration of all peripheral motor neurons secondary to an inherited primary genetic immunodeficiency
(C) Complete and permanent systemic blockage of all voltage-gated calcium channels by excessively produced antigen-specific IgE
(D) The physical and structural occlusion of neuromuscular junctions by massive deposits of insoluble, cross-linked IgM complexes

2002

210. Which process best describes the concept of immunological cross-reactivity?

(A) A mature B cell completely changes its genetic sequence to match a new invading pathogen perfectly
(B) A pathogen actively mutates surface proteins to mimic harmless environmental chemical compounds
(C) An antibody or TCR elicited by one antigen binds to a different, structurally similar antigen
(D) Macrophages rapidly exchange phagocytized contents directly with neighboring helper T lymphocytes

Downloaded from www.pdf.tube

receptors, initiating rapid engulfment of the foreign target. Once internalized into a phagosome, this vesicle fuses with lysosomes containing hydrolytic enzymes and reactive oxygen species. This fusion forms a phagolysosome where the pathogen undergoes highly efficient enzymatic degradation and complete cellular destruction.

19. (D) They contain prominent cytoplasmic granules packed with inflammatory mediators
Mast cells, basophils, and eosinophils are critical granulocytes characterized by the presence of prominent cytoplasmic granules containing abundant pre formed inflammatory mediators. Upon activation by immunological or non immunological triggers, these cells undergo rapid degranulation. This process releases molecules such as histamine, enzymes, and toxic proteins into the tissue microenvironment to coordinate immune responses.

20. (A) Release of major basic protein and peroxidase onto the parasite's surface
Helminth parasites are generally too large to be phagocytosed by immune cells. Eosinophils combat these pathogens by binding to antibody coated surfaces and releasing the toxic contents of their granules directly onto the tegument of the parasite. This granular release includes major basic protein and eosinophil peroxidase, which inflict severe extracellular tissue damage.

21. (C) They circulate continuously in the blood and only enter tissues during active inflammation
Mast cells do not circulate as mature cells in the bloodstream. Instead, they exit the bone marrow as immature progenitors and migrate into peripheral tissues, where they undergo terminal differentiation and remain resident. This strategic tissue localization places them at host environment interfaces to rapidly detect pathogens and initiate immediate inflammatory cellular signaling.

22. (A) Mast cell : Histamine
Mast cells are characterized by highly developed cytoplasmic granules filled with pre formed inflammatory mediators. Among these stored compounds, histamine is the primary and most prominent vasoactive amine. Upon activation and subsequent degranulation, histamine is rapidly discharged into the extracellular space, causing immediate physiological changes such as localized vasodilation and increased vascular permeability.

23. (D) Eosinophils : Exclusively confined to primary lymphoid organs
Eosinophils are not exclusively confined to primary lymphoid organs. In healthy animals, these cells are produced in the bone marrow and circulate briefly in the bloodstream before migrating into tissues, particularly mucosal surfaces of the respiratory, gastrointestinal, and urogenital tracts. Their tissue distribution allows them to respond to mucosal pathogens and allergens.

24. (B) Major Basic Protein (MBP)
Major basic protein is a highly toxic molecule stored in and released by eosinophils, serving as a primary weapon against helminths. Mast cells do not synthesize or store major basic protein. Instead, mast cells release a distinct profile of mediators, including histamine, heparin, and serotonin, to orchestrate localized vascular and inflammatory responses.

25. (B) I, III and IV
Both mast cells and basophils express high affinity Fc receptors for immunoglobulin E on their surface and release histamine upon receptor cross-linking. Mast cells are resident in tissues, whereas basophils are circulating granulocytes that enter tissues during inflammatory responses. Both cell types are key mediators in the development of type I hypersensitivity reactions.

26. (D) Vasodilation and increased vascular permeability
Histamine released during mast cell degranulation binds to specific receptors on vascular smooth muscle and endothelial cells. This binding triggers rapid relaxation of vascular smooth muscle, causing vasodilation, and induces endothelial cell contraction. The resulting endothelial gaps increase vascular permeability, allowing fluid and plasma proteins to escape into surrounding tissues, leading to local edema.

NODIA

27. (A) Binding of the C1 complex to antigen-bound IgG or IgM molecules
The classical pathway is initiated when the C1 complex binds to the Fc portion of antigen bound immunoglobulin G or immunoglobulin M. This binding induces a conformational change in C1, activating its enzymatic subunits to initiate

in determining susceptibility to atopic dermatitis or associated hypersensitivity reactions.

63. (C) Profound, sustained bronchoconstriction and widespread vasodilation causing hypotension
During systemic anaphylaxis, de novo synthesized lipid mediators like leukotrienes and prostaglandins are released alongside histamine. These potent compounds act directly on smooth muscle to induce profound, prolonged bronchoconstriction in the airways. They concurrently cause widespread systemic vasodilation and increased vascular permeability, leading to a rapid drop in blood pressure and circulatory shock.

64. (C) Type I immediate hypersensitivity reaction
The presentation of severe facial swelling, erythema, and intense pruritus within twenty minutes of an insect sting represents a classic Type I immediate hypersensitivity reaction. This rapid response is mediated by pre-existing venom-specific IgE antibodies cross-linking on tissue mast cells, causing immediate degranulation and release of vasoactive histamine into local tissues.

65. (C) Antigen injection → IgG complex formation → Complement activation → Neutrophil infiltration and necrosis
The Arthus reaction is a localized Type III hypersensitivity initiated when antigen is injected into an animal with high levels of circulating IgG. This leads to local immune complex formation, which activates the complement cascade. Active complement fragments then recruit neutrophils, which release destructive lysosomal enzymes, causing local vascular damage, thrombosis, and tissue necrosis.

66. (B) Both are Type I reactions, but anaphylaxis involves systemic shock, while atopy is localized or regional
Systemic anaphylaxis and atopy share the same underlying immunological mechanism, being classified as IgE-mediated Type I hypersensitivity reactions. However, they differ in their clinical distribution. Anaphylaxis is a life-threatening systemic event characterized by generalized mast cell degranulation, vascular collapse, and bronchoconstriction. In contrast, atopy represents a localized, often chronic allergic reaction restricted to target tissues.

67. (D) The pathogenesis is strictly limited to Type II cytotoxic hypersensitivity destroying the respiratory epithelium
The pathogenesis of feline and equine asthma is primarily driven by Type I and Type IV hypersensitivity responses rather than Type II cytotoxic mechanisms. These diseases involve allergen-induced IgE cross-linking, Th2 cell activation, and eosinophilic airway inflammation, which trigger bronchospasm and remodeling. Epithelial damage is a secondary consequence of chronic inflammation, not direct antibody-mediated cytotoxicity.

68. (A) Complement lysis, antibody-dependent cellular cytotoxicity, and opsonization
Type II hypersensitivity damages host cells through antibody-mediated pathways. When IgG or IgM binds to cell surface antigens, it activates the classical complement cascade, leading to direct osmotic lysis or C3b-mediated opsonization for phagocytosis. Alternatively, antibody-coated cells are targeted by effector cells expressing Fc receptors, which destroy them via antibody-dependent cellular cytotoxicity.

69. (C) Natural Killer (NK) cell
Natural Killer cells function as primary effectors in antibody-dependent cellular cytotoxicity. They express the surface receptor CD16, a high-affinity Fc-gamma receptor that binds to the Fc portion of IgG antibodies already attached to target cells. This binding cross-links the receptors, triggering the polarized release of perforin and granzymes to induce target cell apoptosis.

70. (B) A Type II reaction where maternal colostral alloantibodies destroy foal red cells
Neonatal isoerythrolysis in foals is a Type II cytotoxic hypersensitivity reaction. It occurs when a foal inherits a red blood cell antigen from its sire that is absent in the dam. If the dam has been sensitized against this antigen, she secretes specific alloantibodies into her colostrum. Upon ingestion, these antibodies are absorbed and destroy the foal's red cells.

71. (D) A drug hapten binding red cells and eliciting an IgG response that destroys them
Drug-induced cytopenia can occur via a Type II mechanism when a drug acts as a hapten, binding covalently to cell surface proteins on erythrocytes. This newly formed drug-cell

Click Here to Buy book on Amazon

37. (C) Immunocompetence develops in a stepwise manner, recognizing different antigens at progressive gestational stages
Fetal immunocompetence develops gradually throughout gestation rather than occurring all at once. The developing fetus becomes competent to respond to different antigens in a predictable, stepwise chronological order. This maturation of antigen responsiveness correlates with the progressive development and differentiation of specific lymphocyte populations and lymphoid microenvironments in the fetal tissues and organs.

38. (A) Maternal IgG completely suppresses fetal hematopoiesis until after birth
Maternal immunoglobulin G does not suppress fetal hematopoiesis. The development of hematopoiesis in the fetal liver, spleen, and bone marrow is a regulated physiological process essential for producing fetal red and white blood cells. This continuous blood cell generation occurs independently of maternal antibodies, ensuring the fetus possesses functional innate and adaptive immune cells.

39. (D) Early embryonic yolk sac : Site of initial hemopoiesis and stem cell generation
During early embryonic development, the yolk sac serves as the initial anatomical site for hemopoiesis. It is responsible for generating the first primitive hematopoietic stem cells and red blood cells. These precursor cells later migrate to seed the fetal liver, spleen, and bone marrow to establish definitive adult hematopoiesis and initiate immune cell development.

40. (B) Fetal antibody production : Heavily skewed toward the production of IgE
Fetal antibody production is not skewed toward immunoglobulin E. Instead, the primary immunoglobulins synthesized by the fetus are immunoglobulin M, which is the first class expressed by maturing B lymphocytes, and occasionally small amounts of immunoglobulin G. Immunoglobulin E production remains very low during fetal life, only increasing postnatally during parasitic infections or allergic sensitization.

41. (C) Complete anatomical absence of secondary lymphoid organs like lymph nodes
Neonates do not lack secondary lymphoid organs. Lymph nodes, spleen, and mucosal lymphoid tissues are anatomically present at birth, although they are immunologically naive and lack active germinal centers. The relative neonatal immunodeficiency is instead driven by elevated cortisol, naive lymphocyte pools, and down regulated antigen presentation, rather than a lack of physical lymphoid structures.

NODIA

42. (D) Presence of elevated specific IgM antibodies in the pre-suckle serum
Finding elevated antigen specific immunoglobulin M in pre suckle serum indicates active fetal synthesis because maternal antibodies cannot cross the epitheliochorial placenta of mares. Since the foal has not yet ingested colostrum, any immunoglobulins present in its blood must have been generated in utero by the fetal immune system in response to pathogen exposure.

43. (A) Viral antigens in the thymus trigger clonal deletion of specific T-cells during negative selection
Exposure of the bovine fetus to non cytopathic bovine viral diarrhea virus before it reaches full immunocompetence allows viral antigens to enter the developing thymus. The thymus treats these foreign antigens as self proteins during T cell selection. This triggers negative selection and clonal deletion of virus specific T cells, leaving the animal permanently unable to clear the infection.

44. (B) Mummification/abortion if early, immune clearance/survival if late
The outcome of fetal infection is heavily determined by gestational age. Early in gestation, before the development of immune structures, the fetus is highly vulnerable, which often leads to fetal death, mummification, or abortion. Conversely, late gestation exposure occurs when the fetus is immunocompetent, allowing it to mount an active immune response for successful pathogen clearance.

45. (A) Both A and R are true and R is the correct explanation of A.
Domestic ungulates like horses and cattle utilize an epitheliochorial placenta to separate the maternal and fetal circulations. This placenta features six intact tissue layers, which form a highly restrictive physical barrier that completely blocks the transplacental transfer of large maternal immunoglobulins. Consequently,

96. (D) Myasthenia gravis : Post-synaptic acetylcholine membrane receptors
Myasthenia gravis is an organ-specific autoimmune disease targeting post-synaptic acetylcholine receptors at neuromuscular junctions. Autoantibodies bind to these receptors, inducing receptor internalization and complement-mediated destruction of the post-synaptic membrane. This reduces the number of functional receptors, blocking acetylcholine binding and impairing neuromuscular transmission, which manifests as muscle weakness in affected animals.

97. (C) Reversible muscle weakness after exercise due to autoantibody blockade of acetylcholine receptors
Acquired myasthenia gravis is caused by circulating autoantibodies that block and destroy post-synaptic acetylcholine receptors. Clinically, this impairment of neuromuscular signaling leads to progressive, exercise-induced muscle weakness. Resting allows acetylcholine levels to accumulate in the synaptic cleft, temporarily overcoming the competitive receptor blockade and restoring muscle strength, making the weakness characteristically reversible.

98. (B) An autosomal recessive mutation in the DNA-PKcs gene, disrupting V(D)J recombination
Severe Combined Immunodeficiency in Arabian foals is a hereditary, primary congenital immunodeficiency. It is caused by an autosomal recessive mutation in the gene encoding the catalytic subunit of DNA-dependent protein kinase. This mutation disrupts the V(D)J recombination process, preventing the proper rearrangement of antigen receptor genes in both developing T-lymphocytes and B-lymphocytes.

99. (D) Absence of B and T lymphocytes, leading to fatal opportunistic infections as colostral antibodies wane
Foals born with Severe Combined Immunodeficiency are completely unable to produce functional antigen-specific lymphocytes. Consequently, they exhibit a total absence of mature B-lymphocytes and T-lymphocytes in lymphoid organs and blood. While maternal colostral antibodies provide temporary passive protection, once these antibodies decline around two months of age, the foal succumbs to fatal opportunistic infections.

100. (A) Profound lymphopenia → Hypogammaglobulinemia post-weaning → Confirmation of DNA-PKcs mutation via PCR
Diagnosing Severe Combined Immunodeficiency in Arabian foals involves identifying key clinical and genetic markers. The evaluation begins with finding profound lymphopenia in peripheral blood smears. This is followed by documenting severe hypogammaglobulinemia after maternal colostral antibodies have declined. A definitive diagnosis is achieved by confirming the presence of the genetic mutation using polymerase chain reaction testing.

101. (C) Recurrent mucosal infections, including chronic enteritis and respiratory disease
Immunoglobulin A is the primary antibody class responsible for protecting host mucosal surfaces. A selective deficiency of IgA impairs immune exclusion at these barrier zones, leaving mucosal tissues vulnerable to pathogen colonization. Consequently, affected dogs, such as German Shepherds, exhibit a high susceptibility to recurrent mucosal conditions, including chronic enteritis and respiratory tract infections.

102. (A) It causes an absolute neutropenia (lack of neutrophils) in the circulating peripheral blood
Bovine Leukocyte Adhesion Deficiency does not cause neutropenia. Instead, it results in a marked, persistent neutrophilia in the peripheral blood circulation. Because neutrophils lack functional CD18 beta-two integrins, they cannot adhere to the vascular endothelium or migrate into tissues. Consequently, they remain trapped within the bloodstream, unable to exit and combat local infections.

103. (D) Neutrophils cannot exit the bloodstream to reach infection sites due to defective adhesion molecules
In Bovine Leukocyte Adhesion Deficiency, a genetic mutation in the CD18 integrin subunit prevents the expression of functional adhesion molecules on the surface of neutrophils. Although these granulocytes are produced in massive numbers, they cannot bind tightly to the vascular endothelium. This prevents diapedesis, leaving them unable to migrate into tissues to eliminate localized bacterial pathogens.

Downloaded from www.pdf.tube

54. (A) Serum IgG > 800 mg/dL : Adequate passive transfer
In equine veterinary medicine, measuring systemic immunoglobulin G levels is the standard diagnostic tool for assessing passive transfer. A concentration exceeding eight hundred milligrams per deciliter indicates successful passive transfer, associated with a lower risk of neonatal sepsis. Levels below this threshold represent partial or complete failure of passive transfer, requiring veterinary clinical intervention.

55. (B) P-1, Q-4, R-3, S-2
The Gell and Coombs classification system categorizes hypersensitivity into four distinct types based on active mediators. Type I is driven by IgE and mast cells. Type II involves antibodies targeting cell surface antigens. Type III is mediated by soluble immune complexes. Finally, Type IV relies on T lymphocytes and macrophages, functioning independently of antibody classes.

56. (D) Type IV : IgA-dependent mucosal hypersensitivity
Type IV hypersensitivity is uniquely defined as a cell mediated delayed reaction driven exclusively by activated T lymphocytes and macrophages. Unlike the first three categories, Type IV reactions do not involve any antibody classes, making the association with IgA completely incorrect. It typically requires forty-eight to seventy-two hours to manifest following antigen exposure.

57. (C) Type III involves deposition of circulating complexes, while Type II involves antibodies binding directly to cell surfaces
Type II hypersensitivity occurs when antibodies bind directly to fixed antigens on specific cell membranes or tissues. In contrast, Type III hypersensitivity is initiated by the formation of soluble antigen antibody complexes in the bloodstream. These complexes later deposit within vascular walls or filter membranes, initiating complement activation and subsequent neutrophil mediated tissue damage.

58. (D) Histamine-laden basophils
Type IV hypersensitivity relies strictly on T cell mediated pathways, involving $CD4^+$ helper T cells, macrophages, and cytokine signals like interferon gamma. Histamine laden basophils are granulocytes central to antibody dependent immediate Type I hypersensitivity reactions. They do not play an active role in driving the delayed, cell mediated processes characteristic of Type IV immune responses.

59. (A) It requires time for antigen processing, T-cell activation, and the subsequent recruitment of macrophages
The delay in Type IV delayed-type hypersensitivity skin reactions occurs because several complex cellular processes must happen sequentially. Upon intradermal injection, antigen-presenting cells must capture, process, and present the antigen. Memory T lymphocytes then recognize the antigen, activate, secrete chemokines, and recruit circulating macrophages to migrate to the injection site, causing visible swelling.

NODIA

60. (B) Cross-linking of mast cell-bound IgE by a multivalent allergen
Type I hypersensitivity is initiated when a sensitized host is re-exposed to a multivalent allergen. This allergen binds to and cross-links specific immunoglobulin E antibodies already attached to high affinity receptors on tissue mast cells. This cross-linking activates intracellular signaling pathways, triggering immediate degranulation and the rapid release of pre formed vasoactive mediators.

61. (B) Allergen exposure → Th2 activation → IL-4 production → B-cell isotype switch → IgE binding to mast cells
Sensitization begins with initial allergen exposure, promoting helper T-cell differentiation into the Th2 lineage. Activated Th2 cells produce interleukin-4, which instructs B-lymphocytes to undergo isotype switching to produce allergen-specific IgE. These circulating IgE molecules subsequently bind to high-affinity Fc receptors on tissue mast cells, leaving the animal primed for future hypersensitivity reactions.

62. (A) The individual's blood type and erythrocyte surface antigens
Atopic dermatitis is a complex inflammatory skin disease driven by a genetic predisposition to synthesize IgE against environmental allergens, combined with epidermal barrier defects. An animal's blood type and specific erythrocyte surface antigens are completely unrelated to these immunological pathways and play no role

80. (D) The damage invariably leads to massive polyuria and immediate acute renal failure within minutes
Immune-mediated glomerulonephritis is a chronic, progressive disease driven by immune complex deposition in the glomerular basement membrane. This process does not cause acute renal failure within minutes. Instead, the inflammatory damage develops over weeks to months, leading to gradual loss of filtration barrier integrity, persistent protein leakage, and eventual chronic renal insufficiency.

81. (C) Antibody formation against foreign serum over 7-10 days, leading to complexes, complement activation, and vasculitis
Serum sickness is a systemic Type III hypersensitivity that occurs after receiving foreign proteins. Over seven to ten days, the host mounts an antibody response against these antigens. As antibodies are produced, they form soluble complexes with circulating foreign proteins. These complexes deposit in blood vessels and joints, activating complement and causing widespread vasculitis.

82. (B) Presence of intracellular bacteria undergoing binary fission
Immune-mediated polyarthritis is a sterile inflammatory joint disease driven by immune complex deposition in the synovium, not by joint infection. Finding intracellular bacteria undergoing binary fission indicates septic arthritis, which is a direct infectious disease. Septic arthritis requires a different clinical approach, whereas immune-mediated polyarthritis typically presents with sterile, neutrophil-rich joint fluid.

83. (D) Interferon-gamma (IFN-γ)
In Type IV delayed-type hypersensitivity, activated $CD4^+$ helper T-lymphocytes of the Th1 subclass secrete several cytokine signals. Among these, interferon-gamma serves as the primary and most critical cytokine. It acts directly on tissue macrophages, recruiting them to the site of antigen exposure and significantly enhancing their microbicidal activities, phagocytosis, and antigen presentation.

84. (A) Memory Th1 cells must encounter the antigen, secrete active cytokines, and recruit macrophages, which is a slow physiological process
The tuberculin test is a classic delayed hypersensitivity reaction. It requires seventy-two hours because the response relies on cell-mediated pathways rather than pre-formed antibodies. Antigen-presenting cells must process the tuberculin. Memory T-lymphocytes then recognize it, activate, and secrete cytokines to recruit circulating macrophages, which slowly accumulate to form a swelling.

85. (B) I and II
Contact dermatitis represents a Type IV delayed-type hypersensitivity reaction. The offending chemical compound is usually a small molecule called a hapten, which cannot elicit an immune response alone. It must bind covalently to endogenous host skin proteins to form an immunogenic conjugate, which is subsequently processed by dendritic cells to activate T-lymphocytes.

86. (A) By driving the formation of a chronic granuloma composed of macrophages, giant cells, and lymphocytes
When intracellular mycobacteria resist destruction inside macrophages, persistent antigen exposure drives a chronic Type IV hypersensitivity response. T-lymphocytes continuously secrete cytokines that recruit and activate additional macrophages, causing them to fuse into multinucleated giant cells. This chronic inflammatory process forms a structured granuloma that physically walls off and contains the persistent intracellular pathogen.

87. (D) Induction of central and peripheral immunological tolerance to self-antigens
Healthy animals are protected from self-reactivity by immunological tolerance, which is categorized into central and peripheral mechanisms. Central tolerance eliminates autoreactive lymphocyte clones during their development in primary lymphoid organs. Any escaping autoreactive cells are managed in the periphery by mechanisms of peripheral tolerance, including anergy, suppression by regulatory cells, and clonal deletion.

Click Here to Buy book on Flipkart

complex becomes immunogenic, eliciting the production of IgG antibodies against the hapten-protein conjugate. Subsequent antibody binding initiates complement activation or macrophage phagocytosis, resulting in cell destruction.

72. (B) Recipient antibodies binding donor RBC membranes to activate the classical complement cascade
Acute intravascular hemolysis during an incompatible blood transfusion is driven by a Type II cytotoxic reaction. Recipient antibodies rapidly bind to foreign antigens on the donor red blood cell membranes. This antigen-antibody interaction initiates the classical complement cascade, culminating in the assembly of membrane attack complexes that physically perforate the donor cell membranes, causing rapid osmotic lysis.

73. (C) Accelerated splenic macrophage phagocytosis of RBCs (extravascular hemolysis)
In autoimmune hemolytic anemia, autoantibodies and complement proteins coat the surfaces of host erythrocytes. This opsonization is recognized by macrophages in the spleen and liver via surface Fc-gamma and C3b receptors. Rather than lysing within blood vessels, these opsonized cells are phagocytosed and destroyed by splenic macrophages, leading to accelerated extravascular hemolysis.

74. (D) Pre-existing IgG or IgM will bind transfused erythrocytes, triggering complement activation and hemolysis
Previously sensitized dogs possess circulating, antigen-specific immunoglobulin G or immunoglobulin M antibodies against incompatible erythrocyte surface proteins. Upon receiving a mismatched transfusion, these pre-existing antibodies immediately bind the donor red blood cells. This rapid binding triggers the classical complement cascade, causing acute intravascular hemolysis, systemic inflammatory activation, and potentially fatal transfusion reactions.

75. (A) Both A and R are true and R is the correct explanation of A.
The direct Coombs test is the gold standard diagnostic tool for confirming immune-mediated hemolytic anemia in animals. This assay functions by detecting the presence of host antibodies or complement proteins already bound to the surfaces of the patient's red blood cells, using anti-species immunoglobulins to induce visible agglutination of these cells.

76. (B) By activating complement to generate C5a, which recruits neutrophils that release destructive enzymes
When soluble immune complexes deposit within vascular walls or filtering tissues, they activate the complement cascade. This activation generates C5a, a highly potent chemoattractant. C5a recruits circulating neutrophils to the deposition site. Once there, these neutrophils undergo activation and release destructive lysosomal enzymes and reactive oxygen species, causing local tissue inflammation and necrosis.

77. (B) Contact dermatitis to poison ivy : Type II hypersensitivity
Contact dermatitis to poison ivy is a classic Type IV delayed-type hypersensitivity reaction rather than a Type II cytotoxic reaction. The reaction is driven by T-lymphocytes recognizing haptenated skin proteins, resulting in cytokine release and macrophage recruitment. It is completely independent of antibody binding to cells or complement-mediated cell lysis, which defines Type II hypersensitivity.

78. (A) The complexes form at intermediate antigen-antibody ratios, preventing efficient phagocytic clearance
Immune complex deposition is heavily determined by the ratio of antigen to antibody. Under conditions of slight antigen excess, small or intermediate sized soluble complexes form. Unlike large complexes formed at equivalence, these intermediate complexes are not easily recognized by macrophage Fc or complement receptors. Consequently, they escape splenic phagocytosis, persist in circulation, and deposit in tissues.

79. (C) Adenovirus-induced "blue eye" from complex deposition
Canine adenovirus type one infection or vaccination can cause a localized Type III hypersensitivity reaction in the eye, commonly known as blue eye. This condition occurs when soluble viral antigen-antibody complexes deposit within the anterior uvea and corneal endothelium. This deposition activates complement, recruiting neutrophils that cause corneal edema and a characteristic blue appearance.

Downloaded from www.pdf.tube

downstream cleavage of complement proteins C_4 and C2. Free, unbound immunoglobulins cannot initiate this pathway effectively.

28. (B) C3bBb
In the alternative pathway of complement activation, spontaneous hydrolysis of C3 generates C3b, which binds to Factor B on pathogen surfaces. Factor D then cleaves Factor B, releasing the Ba fragment and leaving Bb attached. This forms the active bimolecular complex C3bBb, which functions as the alternative pathway C3 convertase to amplify C3 cleavage.

29. (B) The classical pathway is antibody-dependent, while the alternative pathway functions as an antibody-independent innate defense
The classical complement pathway relies on the adaptive immune response, requiring pre existing antigen specific antibodies to bind target epitopes before C1 can attach and initiate activation. In contrast, the alternative pathway is an innate, antibody independent defense mechanism initiated by the spontaneous hydrolysis of C3 and direct interaction with foreign surfaces.

30. (A) Induction of specific somatic hypermutation in memory B cells
Complement activation during acute infections is a crucial innate defense that leads to pathogen opsonization, membrane attack complex assembly, and inflammatory cell recruitment. It is not involved in inducing somatic hypermutation in memory B lymphocytes. Somatic hypermutation is instead an antigen driven process that takes place inside germinal centers, regulated by follicular helper T lymphocytes.

31. (C) Defective assembly of the cytolytic Membrane Attack Complex (MAC) pore
The terminal step of the complement cascade involves the polymerization of multiple C9 proteins around a core complex of C5b-8. This polymer forms the transmembrane pore of the membrane attack complex, which induces osmotic cell lysis. Lacking functional C9 prevents proper pore assembly, leaving the host unable to lyse pathogens via this specific mechanism.

32. (D) Expression of CD59 and Decay Accelerating Factor (DAF) on host membranes
Host cell membranes express specialized regulatory proteins to protect self tissues from accidental complement mediated damage. Decay accelerating factor rapidly dissociates C3 and C5 convertase complexes, halting the cascade. Concurrently, CD59 binds to the C5b-8 complex, blocking the recruitment and polymerization of C9 proteins, thereby preventing the formation of cytolytic membrane attack complex pores.

33. (C) The C5a fragment acts as a potent anaphylatoxin and neutrophil chemoattractant
Cleavage of complement protein C5 by C5 convertase produces C5b, which initiates terminal pore formation, and C5a, which is released into the microenvironment. C5a acts as a powerful anaphylatoxin, stimulating mast cell degranulation, and functions as a highly potent chemoattractant that directs neutrophils to migrate toward the site of active tissue infection.

34. (B) Cleavage of C5 into C5a and C5b to initiate the terminal pathway
The C5 convertase complex is a multi protein enzyme assembled on cell surfaces during complement activation. Its primary enzymatic function is to bind and cleave the C5 molecule. This cleavage produces C5a, a soluble mediator, and C5b, which binds to the membrane and initiates the non enzymatic terminal pathway of membrane attack complex assembly.

35. (C) I, II and IV
The terminal complement pathway is identical across all pathways, initiating with C5b binding to C6, C7, and C8. This complex inserts into the cell membrane, allowing multiple C9 molecules to polymerize and form a cytolytic pore. Host cells express regulatory proteins to prevent damage, while C3a is an anaphylatoxin rather than a pore component.

36. (A) Appearance of early lymphoid organs and lymphocyte precursors
During gestational development, the earliest milestone of the fetal immune system is the anatomical appearance of primary lymphoid organs and the seeding of lymphoid precursor cells. This structural foundation precedes the functional capacity to undergo immunoglobulin class switching, generate mature memory cells, or execute complex cell mediated reactions, establishing the basis for eventual immunocompetence.

NODIA

88. (B) By triggering apoptosis (negative selection) in developing T-cells that bind self-antigens in the thymus
Central tolerance operates within the thymus through a process known as negative selection. Developing T-lymphocytes are exposed to self-antigens presented by thymic epithelial and dendritic cells. If a T-lymphocyte binds these self-antigens with high affinity, it receives signals that trigger programmed cell death, effectively deleting the potentially autoreactive clone before it matures.

89. (C) A pathogen antigen resembles a host self-antigen, causing the immune response to cross-react with host tissues
Molecular mimicry is a mechanism where a foreign pathogen possesses epitopes that share structural similarity with host self-antigens. When the immune system mounts a response to clear the pathogen, the activated antibodies or T-lymphocytes can cross-react with the resembling host tissues. This cross-reactivity breaks immunological tolerance and initiates autoimmune disease in the host.

90. (D) Inflammation or injury alters self-proteins, revealing hidden antigenic determinants to which the system was not tolerized
Cryptic epitopes are hidden structural determinants on self-proteins that are not normally presented to the immune system. Consequently, developing lymphocytes are never tolerized against them. When tissue injury, enzymatic cleavage, or severe inflammation alters these self-proteins, these cryptic determinants become exposed. Lymphocytes recognize them as foreign, initiating an autoimmune inflammatory attack.

91. (A) An initial immune response broadens to target distinct epitopes on the same or related autoantigens as damage continues
Epitope spreading is a progressive phenomenon observed in chronic autoimmune diseases. As the initial immune response damages host tissues, intracellular proteins are released and processed. This tissue destruction exposes new, distinct epitopes on the same or related self-proteins. Consequently, the autoimmune response broadens to target these newly revealed epitopes, intensifying the chronic inflammatory process.

92. (B) A breakdown in tolerance producing autoantibodies, like ANA, causing systemic immune complex deposition
Systemic Lupus Erythematosus is a multi-system autoimmune disease characterized by a fundamental breakdown in self-tolerance. Affected dogs produce diverse autoantibodies, most notably antinuclear antibodies targeting nuclear components. These autoantibodies form soluble immune complexes with circulating nuclear debris. The subsequent systemic deposition of these complexes in tissue beds drives complement-mediated inflammation and widespread damage.

93. (B) A phagocyte that has ingested nuclear material previously opsonized by antinuclear antibodies from circulating serum
The lupus erythematosus cell is a characteristic cytological finding in Systemic Lupus Erythematosus. It is a mature phagocyte, typically a neutrophil, that has engulfed a large, amorphous extracellular mass of nuclear material. This nuclear material was previously released from damaged cells and opsonized by circulating antinuclear antibodies, facilitating its recognition and subsequent phagocytosis.

94. (A) The vascular deposition of circulating immune complexes followed by complement-mediated inflammation
The multi-systemic lesions observed in canine Systemic Lupus Erythematosus represent Type III hypersensitivity pathology. Soluble antinuclear immune complexes circulate through the bloodstream and deposit in capillaries within joints, renal glomeruli, and the dermis. This local tissue deposition triggers classical complement activation, generating chemoattractants that recruit neutrophils to initiate destructive tissue inflammation and necrosis.

95. (A) I, II and IV
The Pemphigus complex consists of organ-specific autoimmune diseases targeting desmosomal proteins. Autoantibodies binding to these proteins induce acantholysis, destroying cell-to-cell adhesion between keratinocytes. This leads to vesicle and pustule formation. Notably, pemphigus foliaceus is a superficial form targeting upper epidermal desmoglein-1, whereas pemphigus vulgaris is deeper, targeting deeper epidermal desmoglein-3.

Click Here to Buy book on Amazon

these neonates are born agammaglobulinemic and are entirely dependent on colostrum.

46. (B) Endotheliochorial placenta allowing partial transfer (5-10%) of maternal IgG
Carnivores like dogs and cats possess an endotheliochorial placenta, where the maternal vascular endothelium directly contacts the fetal chorion. This anatomical arrangement is less restrictive than epitheliochorial types, allowing a minor portion, roughly five to ten percent, of maternal immunoglobulin G to pass directly into the fetal circulation, though colostrum intake remains essential.

47. (C) Humans transfer IgG across the placenta, whereas pigs transfer virtually no IgG prenatally
Humans possess a highly invasive hemochorial placenta that permits maternal immunoglobulin G to transfer directly into the fetal bloodstream in utero. In contrast, pigs possess a non invasive epitheliochorial placenta with six tissue layers that completely prevent the prenatal transfer of immunoglobulins. Thus, piglets are born without systemic antibodies, relying entirely on colostrum absorption.

48. (D) The direct mixing of maternal and fetal erythrocytes in the chorionic villi
Maternal antibody transfer across a hemochorial placenta is a highly regulated, active process mediated by neonatal Fc receptors on syncytiotrophoblasts. This transport is isotype selective and depends on maternal antibody concentration. Direct mixing of maternal and fetal blood cells does not occur in a healthy placenta and is completely irrelevant to the active transfer of immunoglobulins.

49. (C) It will suffer from agammaglobulinemia and be highly susceptible to septicemia
Due to the impermeable epitheliochorial placenta in cattle, calves are born with virtually no circulating maternal antibodies. Depriving a newborn calf of colostrum prevents the acquisition of essential immunoglobulins, leaving the animal in a state of severe agammaglobulinemia. Without these protective antibodies, the neonate becomes highly vulnerable to rapid, life threatening bacterial septicemia.

50. (B) Non-selective fluid-phase pinocytosis followed by systemic transcytosis
During the first day of life, neonatal enterocytes possess the unique physiological ability to ingest bulk fluids from the intestinal lumen. They absorb colostral immunoglobulins via non selective fluid phase pinocytosis. These internalized macromolecular proteins are then packaged into intracellular vesicles, transported across the cell, and released into the systemic circulation via lymphatic pathways.

51. (D) It stops the unselective uptake of large macromolecules and potential intestinal pathogens
The onset of gut closure at approximately twenty-four hours after birth marks a crucial transition in the neonate. By halting non selective macromolecular pinocytosis, the specialized enterocytes stop absorbing large intact molecules. This physiological change prevents the systemic entry of environmental pathogens, bacterial toxins, and foreign dietary antigens into the bloodstream, establishing an effective mucosal barrier.

52. (A) FPT induces a primary genetic mutation in the neonate's B-lymphocytes, preventing future antibody synthesis
Failure of passive transfer is an acquired condition resulting from inadequate colostral immunoglobulin intake or absorption, not a genetic disorder. It has no effect on the neonate's genome and does not cause genetic mutations in B lymphocytes. Affected animals retain normal genetic capacity to synthesize their own antibodies once their immune system matures postnatally.

53. (C) Intravenous administration of hyperimmune plasma because the gut is closed to oral immunoglobulins
By thirty-six hours of age, the neonatal enterocytes have completed gut closure, preventing the absorption of orally administered immunoglobulins into the systemic circulation. To resolve failure of passive transfer at this stage, immunoglobulin G must be supplied directly into the bloodstream. Intravenous administration of donor plasma bypasses the closed gut, immediately providing protective antibodies.

NODIA

104. (B) Abnormal intracellular granule fusion forming giant lysosomes in phagocytes, impairing chemotaxis and cell killing
Chediak-Higashi syndrome is an autosomal recessive disorder caused by mutations in the lysosomal trafficking regulator gene. This mutation leads to aberrant intracellular granule fusion, resulting in giant lysosomes within neutrophils and other cells. These giant granules impair cellular chemotaxis and disrupt the release of bactericidal enzymes, severely reducing the host's ability to kill phagocytosed pathogens.

105. (A) I and III
Secondary or acquired immunodeficiencies are not genetic and are instead caused by external factors such as viral infections, stress, drugs, or malnutrition. Conversely, primary immunodeficiencies are hereditary. Severe malnutrition can significantly compromise immune function. Pathogens like feline immunodeficiency virus and chronic stress are classic triggers that induce profound secondary immunodeficiencies in animals.

106. (B) By inducing lymphocyte apoptosis and impairing macrophage and neutrophil functions
Acute Bovine Viral Diarrhea Virus infection suppresses the immune system by targeting lymphoid tissue. The virus induces apoptosis of lymphocytes, leading to severe lymphopenia. Concurrently, it impairs the chemotactic, phagocytic, and microbicidal functions of macrophages and neutrophils, reducing host defenses against secondary opportunistic bacterial infections in both young and mature affected cattle.

107. (A) $CD4^+$ T-helper lymphocytes
Feline Immunodeficiency Virus establishes a chronic, progressive infection that leads to an acquired immunodeficiency syndrome. The virus specifically targets and infects $CD4^+$ T-helper lymphocytes, which are central coordinators of the adaptive immune response. Gradual depletion of these cells compromises both humoral and cell-mediated immunity, increasing susceptibility to opportunistic pathogens and systemic secondary diseases.

108. (C) Bursa of Fabricius
Infectious Bursal Disease virus is a highly contagious disease in young chickens. The virus targets and destroys actively dividing IgM-bearing B-lymphocyte precursors within the bursa of Fabricius, which is the primary site of avian B-cell development. This targeted destruction leads to bursal atrophy and severe, long-lasting humoral immunosuppression in affected domestic birds.

109. (D) CDV targets lymphoid tissue, causing lymphopenia and secondary bacterial infections
Canine Distemper Virus exhibits strong tropism for epithelial, nervous, and lymphoid tissues. During the acute phase of infection, the virus replicates extensively within lymphoid tissues, causing widespread depletion of lymphocytes and resulting in severe lymphopenia. This loss of immune surveillance leaves the host highly vulnerable to secondary bacterial respiratory and gastrointestinal infections.

110. (C) Glucocorticoids (e.g., cortisol)
Chronic environmental stress in livestock activates the hypothalamic-pituitary-adrenal axis, resulting in the sustained release of endogenous glucocorticoids such as cortisol. These hormones act as potent systemic immunosuppressive agents by suppressing lymphocyte proliferation, reducing inflammatory cytokine production, and impairing the migration and bactericidal functions of innate immune cells like tissue neutrophils.

111. (B) Down-regulation of pro-inflammatory cytokines and induction of lymphocyte apoptosis
Elevated concentrations of circulating corticosteroids exert broad immunosuppressive actions by binding to intracellular glucocorticoid receptors. This binding down-regulates the transcription of various pro-inflammatory cytokines, including interleukin-1 and tumor necrosis factor. Concurrently, corticosteroids induce apoptosis in sensitive lymphocyte populations and decrease the overall activation of systemic host adaptive immune response pathways and protective mechanisms.

112. (D) Copper deficiency - Generally enhances macrophage phagocytosis and increases overall disease resistance
Copper deficiency does not enhance immune function; instead, it causes significant immunodeficiency. Copper is an essential component of key antioxidant enzymes, such as superoxide dismutase. Inadequate copper levels impair macrophage function, reduce phagocytic

Downloaded from www.pdf.tube

primary immune response, possessing ten antigen-binding sites that make it exceptionally efficient at activating complement. In contrast, IgG is a high-affinity monomer dominating secondary responses, driving systemic opsonization, toxin neutralization, and immunological memory.

148. (D) The presence of antigen-specific memory cells requiring less activation time and undergoing rapid expansion
The primary immune response takes several days to develop because it requires activating rare naïve lymphocytes. Following antigen clearance, a subset of these cells differentiates into long-lived memory T and B lymphocytes. Upon secondary exposure, these pre-existing memory cells require less costimulatory activation and undergo immediate, rapid clonal expansion, producing robust antibody titers within days.

149. (B) Disulfide bonds : Prevent any flexibility in the hinge region to keep the molecule rigid
Disulfide bonds are critical covalent linkages that stabilize the heavy and light chains of immunoglobulins. However, they do not render the hinge region rigid. The hinge region possesses significant molecular flexibility, which allows the antigen-binding arms to move and adjust, facilitating binding to epitopes spaced at varying distances on target pathogen surfaces.

150. (C) It acts as the primary site for macrophage-mediated extravascular phagocytosis of opsonized erythrocytes
During a Type II hypersensitivity reaction against erythrocytes, the spleen serves as the primary site of destruction. Erythrocytes opsonized with IgG or complement fragments pass through the splenic red pulp, where resident macrophages recognize them via Fc and complement receptors. This interaction triggers extravascular phagocytosis and destruction, leading to progressive hemolytic anemia.

151. (D) Synthesized by plasma cells → binds basolateral poly-Ig receptor → transcytosed and released into lumen
Secretory IgA is synthesized as a dimer by plasma cells residing in the lamina propria. To reach the lumen, dimeric IgA binds to the polymeric immunoglobulin receptor on the basolateral membrane of enterocytes. The receptor-IgA complex is transcytosed through the cell, where the receptor is cleaved, releasing IgA with its protective secretory component.

152. (A) MHC Class I presents endogenous peptides to $CD8^+$ T cells; MHC Class II presents exogenous peptides to $CD4^+$ T cells
Major Histocompatibility Complex molecules display distinct presentation pathways. MHC Class I molecules are expressed on all nucleated cells and present endogenous antigens, such as viral proteins, to $CD8^+$ cytotoxic T cells. In contrast, MHC Class II molecules are expressed on professional antigen-presenting cells and present exogenous antigens to $CD4^+$ T helper cells directly.

153. (B) Antinuclear antibodies (ANA)
Systemic Lupus Erythematosus is a complex, multi-system autoimmune disease. The definitive diagnosis of this condition relies heavily on the detection of antinuclear antibodies in serum. These autoantibodies target intracellular nucleic acids and nucleoproteins. Their presence, combined with characteristic clinical signs like glomerulonephritis, polyarthritis, and dermatological lesions, confirms the diagnosis in affected dogs.

154. (B) Nickel acts as a hapten, binding skin proteins to trigger a Th1-mediated delayed hypersensitivity response
Allergic contact dermatitis is a classic Type IV hypersensitivity reaction. Small reactive molecules, such as nickel, function as haptens. They penetrate the epidermis and covalently bind to endogenous skin proteins. Langerhans cells process these modified proteins and present them to T lymphocytes, causing sensitization and subsequent inflammatory reactions upon future skin re-exposure.

NODIA

155. (C) II and III
Innate immunity represents the first line of host defense. Unlike adaptive immunity, it lacks highly specific immunological memory. Instead, it relies on germline-encoded pattern-recognition receptors, such as Toll-like receptors, to rapidly detect conserved pathogen-associated molecular patterns. This system comprises physical barriers, phagocytic cells like macrophages, and humoral components like the complement system.

121. (B) C3b and Bb
The alternative complement pathway is initiated by the spontaneous hydrolysis of C3 in circulation. Once hydrolyzed, C3b binds to Factor B, which is subsequently cleaved by Factor D to release Ba. This cleavage leaves the active C3bBb complex bound to the target surface, serving as the functional C3 convertase of this pathway.

122. (B) Coating a pathogen with IgG or C3b to enhance phagocytosis by macrophages and neutrophils
Opsonization is a critical biological process that enhances the efficiency of phagocytosis. It involves coating a foreign pathogen or antigen with specific proteins, known as opsonins, such as IgG antibodies or C3b complement fragments. Phagocytic cells, including macrophages and neutrophils, possess specific receptors that bind these opsonins, facilitating rapid binding, internalization, and subsequent pathogen destruction.

123. (A) They require prior sensitization and antigen-specific TCR binding to kill targets
Natural Killer cells are key cellular components of the innate immune system. Unlike T cells, they do not express TCRs and do not require prior exposure or MHC-restricted antigen presentation to identify and destroy abnormal cells. Instead, they use a balance of activating and inhibiting receptors to recognize and kill virally infected or neoplastic host cells.

124. (C) Tissue infiltration by eosinophils and neutrophils, recruited by early-phase mediators
The late-phase response of a Type I hypersensitivity reaction occurs several hours after the initial allergen exposure. While the early phase is driven by pre-formed histamine from mast cells, the late phase is characterized by a significant influx of inflammatory cells, particularly eosinophils and neutrophils, recruited by cytokines and chemokines synthesized during the early activation.

125. (D) P-2, Q-1, R-3, S-4
Mature T and B lymphocytes exhibit distinct surface markers and perform unique immunological functions. Cytotoxic T cells express CD8 and kill infected cells directly. Helper T cells express CD4 and secrete cytokines to guide other cells. Regulatory T cells are defined by FoxP3 expression and suppress responses, whereas B cells express immunoglobulins and differentiate into antibody-secreting plasma cells.

126. (C) Clonal expansion and proliferation of activated T-lymphocytes
Interleukin-2 is a critical autocrine and paracrine growth factor secreted primarily by activated T lymphocytes. It binds to high-affinity receptors on T cells to stimulate clonal expansion, survival, and cell-cycle progression following antigen encounter. A severe deficiency in interleukin-2 production results in a profound failure of T-cell proliferation and general adaptive immune response impairment.

127. (B) Autoimmune Hemolytic Anemia : Type II Hypersensitivity
Autoimmune hemolytic anemia is a classic example of a Type II hypersensitivity reaction. In this condition, host antibodies target and bind directly to specific antigens located on the membranes of circulating erythrocytes. This binding triggers the activation of the classical complement cascade, leading to intravascular hemolysis or extravascular phagocytosis of erythrocytes within the spleen and liver.

128. (D) Allergen
An allergen is defined as an otherwise harmless environmental antigen that elicits an inappropriate and exaggerated Type I hypersensitivity response in sensitized individuals. Upon re-exposure, the allergen binds to and cross-links specific IgE antibodies bound to the surfaces of mast cells and basophils, triggering immediate degranulation and release of vasoactive mediators.

129. (A) It elicits a Th1 response, releasing cytokines that recruit macrophages and cause induration
The tuberculin skin test utilizes purified protein derivative from Mycobacterium bovis to detect sensitized animals. This diagnostic assay relies on a Type IV delayed-type hypersensitivity reaction. Injection of the antigen elicits a local Th1-mediated response, leading to the secretion of cytokines that recruit macrophages, culminating in visible tissue induration at forty-eight to seventy-two hours.

130. (C) Within the first 6 to 12 hours of life
Maximum absorption of colostral immunoglobulins occurs during the first few hours after birth, when specialized neonatal enterocytes are capable of non-specific

Click Here to Buy book on Flipkart

high-affinity Fc receptors on tissue mast cells and circulating basophils.

182. (C) Serving as an essential cofactor for enzymes required for lymphocyte proliferation and thymic function
Dietary zinc deficiency results in a severe secondary immunodeficiency due to its essential metabolic roles. Zinc is an indispensable cofactor for over three hundred enzymes, particularly those regulating nucleic acid synthesis and cell division. Inadequate zinc levels impair lymphocyte proliferation, trigger rapid thymic atrophy, and suppress both cell-mediated and humoral immune responses.

183. (D) The animal develops signs of disease only in extreme old age after a lifetime of good health
Primary immunodeficiencies are congenital disorders resulting from genetic mutations. These conditions typically manifest in young animals shortly after maternal colostral antibody levels decline, often presenting as severe, recurrent, or opportunistic infections that fail to respond to standard treatment. Manifesting only in geriatric animals after a lifetime of healthy function is inconsistent with a congenital defect.

184. (A) Structural similarity between a pathogen epitope and a host antigen causes lymphocytes to cross-react
Molecular mimicry is a key mechanism driving the pathogenesis of autoimmunity. It relies on the structural similarity between epitopes of an infectious pathogen and host self-antigens. When the immune system mounts a response against the pathogen, activated T and B lymphocytes cross-react with these structurally homologous self-antigens, leading to progressive autoantibody production and tissue damage.

185. (A) Both A and R are true and R is the correct explanation of A.
Measurement of total serum protein via refractometry is a valuable and rapid clinical screening tool for evaluating colostral transfer in foals. Maternal immunoglobulins, primarily IgG, are absorbed intact across the gut, constituting the major component of blood globulins. Successful transfer results in a significant, measurable rise in total serum protein from the pre-suckle baseline.

186. (B) Presence of giant fused lysosomal granules in neutrophils and melanocytes, impairing killing and causing albinism
Chediak-Higashi syndrome is an autosomal recessive disorder caused by a mutation in the LYST gene, which regulates lysosomal trafficking. This mutation results in the formation of giant, non-functional fused lysosomal granules in neutrophils and melanocytes. Consequently, affected animals exhibit impaired intracellular bactericidal activity, bleeding tendencies, and partial albinism due to abnormal melanin distribution.

NODIA

187. (A) Ingestion of colostrum containing maternal alloantibodies, induced by a specific BVDV vaccine, that target and destroy neonatal leukocyte and megakaryocyte antigens
Bovine Neonatal Pancytopenia is an alloimmune disease caused by maternal colostral antibodies. Cows vaccinated with a specific BVDV vaccine developed high-affinity alloantibodies targeting MHC Class I molecules present on neonatal tissues. When the calf ingests this colostrum, these maternal antibodies are absorbed and bind to neonatal leukocytes and megakaryocytes, destroying bone marrow progenitor cells.

188. (C) It heavily relies on the massive deposition of circulating antigen-antibody complexes into the tissue capillary beds
Type IV delayed-type hypersensitivity is a purely cell-mediated immune reaction that does not involve antibody production or deposition. Instead, it is mediated by sensitized T lymphocytes, which recruit and activate macrophages to eliminate persistent intracellular pathogens. Associating Type IV hypersensitivity with the deposition of circulating immune complexes in capillary beds is mechanistically incorrect.

189. (D) Foal inherits sire's RBC antigen → maternal IgG is produced against it and transferred via colostrum → causes complement hemolysis of foal's RBCs
Neonatal isoerythrolysis in foals occurs when a newborn inherits a specific red blood cell antigen from its sire that is absent in the dam. If the dam is sensitized against this antigen, she produces specific IgG antibodies which are transferred to the foal via colostrum. Upon ingestion, these antibodies bind to neonatal erythrocytes, triggering complement-mediated hemolysis.

molecule expression and enhancing the subsequent adaptive immune response.

139. (A) Presentation of endogenous antigens strictly on MHC Class I
Macrophages are professional antigen-presenting cells that process and present exogenous antigens on MHC Class II molecules to $CD4^+$ T helper cells. They also produce acute-phase cytokines and phagocytize cellular debris. In contrast, presentation of endogenous antigens on MHC Class I is a general function of nucleated cells, not a distinguishing characteristic of mature macrophages.

140. (C) Deposition of antigen-antibody complexes in the uvea, causing Type III hypersensitivity and complement activation
The "blue eye" clinical phenomenon is a localized immune-mediated reaction observed during recovery from canine adenovirus type 1 infection or following administration of modified-live vaccines. This corneal edema represents a classic Type III hypersensitivity, driven by the deposition of soluble viral antigen-antibody complexes in the uvea, triggering complement activation, neutrophil recruitment, and corneal endothelial damage.

141. (D) The immunoglobulin concentration in colostrum continuously increases for the first two weeks of lactation
Colostrum is a transient secretory product produced by the mammary gland immediately post-parturition. It differs significantly from mature milk by possessing extremely high levels of maternal immunoglobulins, cytokines, and leukocytes. The concentration of immunoglobulins does not increase over time; instead, it rapidly declines within hours of lactation as the secretion transitions to mature milk.

142. (C) The bone marrow
Central tolerance in B-lymphocyte development is a crucial safety mechanism designed to eliminate self-reactive cells before they mature. In most domestic mammals, this selective process occurs primarily within the bone marrow. Developing B cells that encounter self-antigen with high affinity are deleted via apoptosis or undergo receptor editing to change their antigen specificity.

143. (B) CD3 : Forms a signaling complex with the T-cell receptor on mature T cells
CD3 is a highly conserved multi-subunit protein complex that associates non-covalently with the T-cell receptor on all mature T lymphocytes. The primary function of CD3 is to transduce the intracellular biochemical signals required for T-cell activation following the specific binding of the T-cell receptor to its cognate peptide-MHC ligand complex in lymphoid tissues.

144. (D) Apoptosis of short-lived effector lymphocytes, contracting the response to leave memory cells
Resolution of acute inflammation requires active contraction of the immune response once the pathogen is cleared. To restore physiological homeostasis and prevent self-inflicted tissue damage, short-lived effector lymphocytes undergo programmed cell death, or apoptosis. This depletion removes the majority of active immune cells, leaving a small pool of long-lived memory lymphocytes behind in the host.

145. (C) A is true but R is false.
Fresh frozen plasma administration is a highly effective treatment for failure of passive transfer in foals older than twenty-four hours. At this age, specialized enterocytes have been replaced, making oral absorption of immunoglobulins impossible. Intravenous plasma delivery bypasses the closed gut, directly supplying protective maternal antibodies to prevent systemic neonatal septicemia and death.

146. (C) Failure of peripheral tolerance, causing lymphoproliferation and severe autoimmune disease
The Fas-Fas ligand (FasL) pathway is an essential molecular mechanism for maintaining peripheral immunological tolerance. This pathway induces apoptosis in self-reactive lymphocytes and terminates active immune responses. A genetic mutation disrupting this pathway prevents the normal elimination of redundant lymphocytes, leading to uncontrolled lymphoproliferation, systemic autoantibody production, and severe multi-organ autoimmune diseases.

NODIA

147. (A) IgM mediates efficient complement activation; IgG is the main isotype for systemic opsonization and memory
IgM and IgG serve distinct biological roles within the humoral immune response. IgM is synthesized as a large, low-affinity pentamer during the

macromolecular pinocytosis. This absorption capacity begins declining rapidly, with gut closure typically complete within twenty-four hours. Therefore, ingesting colostrum within the first six to twelve hours of life is critical for preventing passive transfer failure.

131. (A) The passive transfer of maternal antibodies, primarily IgG, ingested by the neonate
Colostral immunity refers to the critical process of transferring passive immunity from dam to offspring. Because the epitheliochorial and syndesmochorial placentas of livestock prevent prenatal transfer of immunoglobulins, neonates are born agammaglobulinemic. Ingestion of maternal colostrum rich in IgG and other antibodies provides essential systemic and mucosal protection before active neonatal immune development.

132. (D) A small, non-immunogenic molecule that elicits an immune response only when conjugated to a carrier protein
A hapten is a small, non-immunogenic molecule that possesses antigenic reactivity but is incapable of independently eliciting an immune response. Haptens lack the molecular complexity needed to activate T helper cells. However, when a hapten covalently binds to a larger host carrier protein, the resulting hapten-protein conjugate becomes immunogenic, triggering specific adaptive immune reactions.

133. (B) Bursa of Fabricius : Primary site of T cell maturation in birds
Primary lymphoid organs are sites of lymphocyte production and antigen-independent maturation. The thymus is the primary site of T-cell maturation in all birds and mammals, whereas the bursa of Fabricius is the specialized primary lymphoid organ dedicated solely to B-lymphocyte development in birds. Associating the bursa of Fabricius with avian T-cell maturation is incorrect.

134. (C) Histamine causes systemic vasodilation and increased vascular permeability, reducing peripheral resistance
During systemic anaphylaxis, rapid and widespread degranulation of mast cells and basophils occurs, releasing massive quantities of histamine into circulation. Histamine binds to H1 receptors, causing profound systemic vasodilation and significantly increasing microvascular permeability. This fluid shift out of the vascular space reduces peripheral resistance and venous return, causing severe, life-threatening arterial hypotension.

135. (D) P-2, Q-1, R-4, S-3
Specialized antigen-presenting cells occupy distinct anatomical compartments tailored to local immune surveillance. Langerhans cells are dendritic cells residing in the epidermis. Kupffer cells are resident macrophages located in hepatic sinusoids. Microglia represent the primary myeloid cell population within the central nervous system, and follicular dendritic cells reside within lymph node follicles to present native antigen.

136. (A) Pathogen-specific acquired immunotolerance combined with increased risk for secondary infections
In utero infection of a bovine fetus with non-cytopathic Bovine Viral Diarrhea Virus prior to the development of immunocompetence results in persistent infection. The fetal immune system recognizes the viral proteins as self, establishing lifelong immunotolerance. These persistently infected animals shed massive viral loads and have compromised immune functions, increasing susceptibility to secondary infections.

137. (B) Maternal IgG binds vaccine antigens, preventing active immunization and causing vaccine failure if given too early
Maternal antibody interference is a major cause of early vaccine failure in neonates. Circulating maternal IgG, acquired via colostrum, binds and neutralizes vaccine antigens when administered too early. This rapid clearance prevents the vaccine from interacting with and activating the neonate's naïve lymphocytes, thereby blocking the development of active, protective endogenous immunity.

138. (B) Creating an antigen depot effect and stimulating innate receptors to increase APC activation
Adjuvants are essential formulation components added to killed vaccines to enhance their immunogenicity. They function primarily by creating a localized antigen depot, slowing antigen release, and prolonging presentation to immune cells. Additionally, adjuvants activate pattern recognition receptors on local antigen-presenting cells, stimulating costimulatory

Downloaded from www.pdf.tube

156. (C) An immediate allergic wheal and flare reaction to flea bites
Severe Combined Immunodeficiency is a profound congenital primary immunodeficiency characterized by the absolute failure of both T and B lymphocyte development. Affected animals exhibit severe, life-threatening lymphopenia, recurrent infections, and lymphoid hypoplasia. Allergic immediate reactions, which depend on functional B cells, IgE, and helper T cells, are completely unrelated to this deficiency.

157. (D) Inability of IgM to form pentamers and IgA to form dimers, severely impairing mucosal defense
The joining (J) chain is a small polypeptide crucial for the polymerization of secretory immunoglobulins. It facilitates the assembly of monomeric units into pentameric IgM and dimeric IgA. Blocking J-chain function prevents the formation of these polymeric structures, which severely impairs mucosal immune defense and diminishes complement activation efficiency in the host.

158. (C) C-reactive protein : Acts as the primary somatic hypermutation enzyme in memory B cells
C-reactive protein is an acute-phase protein synthesized by the liver in response to inflammatory cytokines. It binds to phosphocholine on dying host cells and foreign microbes, promoting opsonization and complement activation. It is completely unrelated to somatic hypermutation, which is an antigen-driven enzymatically mediated process occurring inside germinal center B cells.

159. (B) Destruction of multicellular helminths and participation in late-phase allergic reactions
Eosinophils are specialized granulocytes that play a major role in host defense against multicellular parasites, particularly helminths. They release cytotoxic granule contents, including major basic protein and eosinophil cationic protein, directly onto the parasite surface. Additionally, eosinophils participate in late-phase allergic responses by releasing inflammatory mediators and modulating tissue reactions.

160. (D) C1q binds Fc → C1r/s activated → C_4/C2 cleaved → C3 convertase formed → C3 cleaved → C5 convertase formed
The classical complement pathway is initiated when C1q binds to the Fc portion of antigen-bound IgG or IgM. This binding activates C1r and C1s, which cleave C_4 and C2 to form the C3 convertase. The convertase cleaves C3 into C3b and C3a, which subsequently recruits C5 to form the terminal membrane attack complex.

161. (A) The occurrence of affinity maturation and subsequent production of high-affinity IgG antibodies
Somatic hypermutation introduces random point mutations into the variable regions of immunoglobulin genes in germinal center B cells. This genetic diversification is the prerequisite for affinity maturation. B cells expressing mutated antibodies with increased affinity for the presented antigen are selected to survive, leading to the production of highly effective antibodies.

162. (C) A single vaccination at 4 weeks of age guarantees permanent immunity for all puppies, regardless of maternal antibody levels
Neonatal immunization is strongly influenced by maternal antibody levels. High levels of maternal antibodies block successful vaccination, but these decline over time. Due to individual variation, puppies in a single litter reach the window of susceptibility at different times. Therefore, a single vaccine dose given at four weeks cannot guarantee permanent immunity.

NODIA

163. (A) Subcutaneous immune complex formation with circulating IgG, activating complement and neutrophils
The Arthus reaction is a localized Type III hypersensitivity. It occurs when a soluble antigen is injected intradermally or subcutaneously into an animal with high levels of circulating specific IgG. This leads to local immune complex formation, classical complement activation, and C5a production, which recruits neutrophils that release destructive enzymes and cause local necrosis.

164. (D) Type II hypersensitivity
Both autoimmune hemolytic anemia and neonatal isoerythrolysis are mediated by Type II hypersensitivity. These conditions involve cytotoxic antibodies binding directly to surface antigens on host red blood cells. The antibody-

activity, and decrease neutrophil microbicidal ability, leading to lower overall disease resistance in affected domestic livestock populations.

113. (A) BLAD is an irreversible genetic defect, while stress is an acquired and reversible state
Primary immunodeficiencies, such as Bovine Leukocyte Adhesion Deficiency, are inherited genetic mutations that result in permanent and irreversible defects in specific arms of the immune system. In contrast, secondary immunodeficiencies, such as corticosteroid-induced stress, are acquired, temporary, and generally reversible once the primary stressor or underlying disease state is resolved completely.

114. (C) Impaired neutrophil bactericidal activity due to oxidative damage, increasing clinical mastitis risk
Vitamin E and selenium function synergistically as essential antioxidants that protect cellular membranes from oxidative damage. During the respiratory burst, neutrophils generate large quantities of reactive oxygen species to kill phagocytosed bacteria. A deficiency in these nutrients prevents adequate neutralization of these radicals, causing self-inflicted cell damage, impairing neutrophil function, and elevating clinical disease risk.

115. (A) Both A and R are true and R is the correct explanation of A.
Aflatoxins present in contaminated animal feeds are potent immunosuppressants in domestic livestock and poultry. These mycotoxins inhibit protein synthesis by interfering with RNA transcription, which directly suppresses cell-mediated immune responses, macrophage phagocytosis, and the production of specific humoral antibodies. Consequently, affected animals exhibit significantly increased susceptibility to various infectious bacterial and viral diseases.

116. (D) Fetal lymphoid development : Humoral immunity completely matures before innate immunity starts
The ontogeny of the immune system follows a highly structured, developmental timeline. Innate immune defenses, including physical barriers, phagocytic cells, and complement pathways, develop and mature before the adaptive humoral and cell-mediated immune responses are fully functional. Thus, claiming that humoral immunity completely matures before innate immunity begins is developmentally incorrect in animals.

117. (B) They bind target antigens but fail to cross-link them without adding an anti-species globulin
Complete antibodies, such as pentameric IgM, have multiple antigen-binding sites and can easily cross-link particulate antigens to cause visible agglutination. In contrast, incomplete antibodies, which are often IgG monomers, bind to target antigens but are structurally unable to bridge the gap between cells, requiring the addition of an anti-species globulin to achieve visible agglutination.

118. (C) Basophils
Type IV delayed-type hypersensitivity is a classic cell-mediated immune reaction that does not involve antibodies or immediate-release vasoactive amines. It is driven by sensitized $CD4^+$ T-helper 1 cells releasing cytokines that recruit and activate macrophages and epithelioid cells. Basophils are primarily associated with Type I immediate hypersensitivities and are not major effectors in this delayed pathway.

119. (D) Colostrum provides both systemic IgG and localized secretory IgA protecting the neonatal gut
Colostrum provides comprehensive immunological protection to the neonate compared to transplacental transfer alone. It contains high concentrations of IgG, which are absorbed systemic-wide to prevent septicemia. Additionally, colostrum provides a continuous supply of secretory IgA, which remains in the intestinal lumen to neutralize local pathogens, prevent attachment, and physically protect the mucosal lining.

120. (A) Reduced efficacy of prophylactic vaccines due to impaired immune response capacity
When an animal develops a secondary immunodeficiency due to a chronic viral infection, the function of its adaptive immune system becomes severely compromised. This impairment directly reduces the animal's ability to mount an appropriate, protective immune response following the administration of prophylactic vaccines, resulting in vaccine failure and continued susceptibility to target pathogens.

Click Here to Buy book on Amazon

173. (D) The immune response expands from targeting a single self-epitope to attacking additional self-epitopes as tissue damage progresses
Epitope spreading refers to the process during a chronic autoimmune response where tissue damage leads to the release of previously hidden self-antigens. The immune response subsequently expands from targeting the initial single self-epitope to attacking these newly exposed, distinct self-epitopes. This expansion perpetuates the inflammatory cycle and exacerbates multi-organ autoimmune pathology.

174. (C) It binds dimeric IgA in the lamina propria, transporting it across epithelial cells to mucosal surfaces
The polymeric immunoglobulin receptor (poly-Ig receptor) is expressed on the basolateral surface of mucosal epithelial cells. Its primary role is to bind dimeric IgA produced by plasma cells in the lamina propria. Once bound, the receptor-IgA complex is transcytosed to the apical membrane, protecting mucosal surfaces from pathogen colonization and adherence in animals.

175. (A) I, II, III and IV
Canine passive transfer of immunity combines minor placental transfer with major colostral absorption. Dogs transfer five to ten percent of maternal IgG via the endotheliochorial placenta, while the remaining ninety percent must be absorbed via colostrum in the first day of life. Gut closure ends macromolecular absorption, and high titers can interfere with early vaccinations.

176. (D) Failure to transport endogenous peptides into the endoplasmic reticulum, preventing MHC Class I expression
The Transporter associated with Antigen Processing (TAP) is critical for MHC Class I antigen presentation. TAP transports cytosolic peptides, derived from proteasomal degradation of endogenous proteins, into the endoplasmic reticulum. Knocking out TAP prevents these peptides from loading onto MHC Class I molecules, blocking their expression on the cell surface.

177. (A) An intrinsic host molecule that becomes the target of an aberrant autoimmune response
An autoantigen is an endogenous, normal constituent of the host's own cells or tissues that becomes aberrantly targeted by the immune system. Under physiological conditions, central and peripheral tolerance mechanisms prevent responses against self-molecules. In autoimmune diseases, tolerance breaks down, leading to the generation of autoantibodies and pathogenic cell-mediated attacks.

178. (C) Absence of functional regulatory T cells, causing uninhibited multi-organ autoimmunity
The FOXP3 gene is the master transcriptional regulator required for the development and suppressive function of $CD4^{+}CD25+$ regulatory T cells. A genetic mutation in FOXP3 leads to an absolute absence of functional regulatory T cells. Without regulatory oversight, self-reactive T cells undergo uncontrolled expansion, causing severe multi-organ autoimmune diseases in affected animals.

NODIA

179. (A) The MAC relies primarily on the structural scaffolding of circulating IgE to hold it against the cell membrane
The Membrane Attack Complex is a multi-protein pore assembled during the terminal pathway of the complement cascade. It is formed by the sequential integration of C5b, C6, C7, C8, and multiple C9 molecules into the target cell membrane. The MAC does not rely on IgE for structural scaffolding or membrane attachment in vivo.

180. (D) Symptoms arise 7-14 days later as host IgG forms soluble complexes with remaining foreign proteins
Serum sickness is a classic transient Type III hypersensitivity reaction. It occurs seven to fourteen days after administering large quantities of a foreign protein, such as equine antitoxin. During this period, the host synthesizes specific IgG antibodies that bind to the remaining circulating foreign antigens, forming soluble immune complexes that deposit in capillary walls.

181. (B) IgG dominates systemic opsonization and memory, while IgE is bound to mast cells for parasite and allergic defense
IgG and IgE have distinct functional roles in veterinary immunology. IgG is the most abundant immunoglobulin in circulation, dominating systemic defense through opsonization, toxin neutralization, and long-term memory. Conversely, IgE is present in extremely low concentrations in serum because it is bound to

coated cells are subsequently destroyed either via intravascular complement-mediated lysis or extravascular phagocytosis by resident macrophages in the spleen and liver.

165. (B) II only
Systemic Lupus Erythematosus is defined as a non-organ-specific, systemic autoimmune disease that affects multiple organs and tissues. Autoantibodies in this condition target ubiquitous nuclear antigens, leading to widespread vascular, joint, and renal lesions. It is incorrect to categorize it as an organ-specific disease restricted only to the thyroid gland in affected animals.

166. (C) The directional release of perforin and granzymes into the immunological synapse with the target cell
Cytotoxic $CD8^+$ T cells destroy infected host cells through the targeted release of lytic granules containing perforin and granzymes. To protect themselves, T cells establish a highly focused immunological synapse with the target cell. Perforin assembles exclusively in the target membrane, enabling granzymes to enter and trigger apoptosis without injuring the cytotoxic cell.

167. (D) Interleukin-10 : Exerts broadly immunosuppressive and anti-inflammatory effects
Interleukin-10 is a potent anti-inflammatory cytokine produced by regulatory T cells, helper T cells, and macrophages. It plays a vital role in preventing immunopathology by inhibiting the expression of MHC Class II and costimulatory molecules on antigen-presenting cells, reducing pro-inflammatory cytokine synthesis, and suppressing general adaptive cell-mediated immune responses in the animal.

168. (A) Induce long-lived protective memory responses against severe pathogens as maternal antibodies decline
The primary immunological rationale for a core, multivalent vaccination strategy in young animals is to safely induce long-lived adaptive protective immune memory. As maternal colostral antibodies decline, the puppy or kitten becomes vulnerable to virulent pathogens. Structured vaccination stimulates active humoral and cellular defenses, bridging the gap to keep the animal protected from disease.

169. (B) Primary organs generate and mature lymphocytes, while secondary organs are where they undergo activation
Primary lymphoid organs, such as the thymus and bone marrow, generate lymphocytes from progenitor cells and oversee their antigen-independent maturation. Secondary lymphoid organs, including the spleen, lymph nodes, and mucosal-associated lymphoid tissues, serve as specialized anatomical sites where mature lymphocytes migrate, encounter specific foreign antigens, and undergo activation and clonal expansion.

170. (B) The virus depletes $CD4^+$ helper T cells, crippling both humoral and cell-mediated adaptive immune responses
Feline Immunodeficiency Virus targets, infects, and replicates within $CD4^+$ T-helper cells, macrophages, and dendritic cells. The gradual, progressive depletion of $CD4^+$ T-lymphocytes cripples the host immune system. Since T-helper cells are critical for activating $CD8^+$ cytotoxic T cells and stimulating B-cell antibody production, their loss causes severe acquired immunodeficiency in cats.

171. (A) Bovine Leukocyte Adhesion Deficiency (BLAD)
Bovine Leukocyte Adhesion Deficiency is an autosomal recessive genetic defect in the CD18 subunit of integrins. This defect prevents neutrophils from adhering to vascular endothelial cells, blocking extravasation into inflamed tissues. Consequently, affected calves present with marked peripheral neutrophilia but have an absolute lack of neutrophils within localized sites of bacterial infection.

172. (C) Type IV : Free-floating soluble histamine directly attacking tissue
Type IV hypersensitivity is a delayed, cell-mediated reaction that relies on sensitized T lymphocytes and activated macrophages rather than humoral antibodies. It does not involve free-floating soluble histamine, which is a key vasoactive amine pre-formed and released from mast cells and basophils primarily during IgE-mediated Type I immediate hypersensitivity reactions.

Downloaded from www.pdf.tube

190. (A) II, IV, I, III
The pathogenesis of Systemic Lupus Erythematosus follows a structured sequence. Initially, there is a fundamental breakdown of central and/or peripheral immunological self-tolerance. This failure leads to the activation of autoreactive lymphocytes and production of high-affinity antinuclear autoantibodies. These autoantibodies form soluble immune complexes that deposit in capillary beds, triggering complement activation and leukocyte infiltration.

191. (B) Histamine : Profound bronchoconstriction and massive vasodilation
Histamine is a key pre-formed vasoactive amine stored in high concentrations within the secretory granules of mast cells and basophils. During an immediate Type I anaphylactic reaction, massive degranulation releases histamine into circulation. This mediator binds to H1 receptors on smooth muscle and endothelial cells, causing profound bronchoconstriction and widespread peripheral vasodilation.

192. (D) Neutrophils are strictly the most highly efficient and exclusive APCs found within the germinal centers of lymph nodes
Professional antigen-presenting cells are specialized leukocytes that capture, process, and present exogenous antigens to T lymphocytes. This group includes dendritic cells, macrophages, and B cells. In contrast, neutrophils are highly efficient phagocytes specializing in the immediate destruction of bacterial pathogens, but they are not classified as professional antigen-presenting cells within lymphoid follicles.

193. (A) Severe cytokine down-regulation, lymphocyte suppression, and impaired cellular immunity, increasing susceptibility to infection
Prolonged environmental or physiological stress stimulates the sustained secretion of endogenous cortisol in livestock. High levels of circulating corticosteroids act as potent immunosuppressants by down-regulating the transcription of key pro-inflammatory cytokines, suppressing lymphocyte proliferation, and impairing both cell-mediated and humoral immune pathways. This compromised state significantly increases susceptibility to opportunistic infectious diseases.

194. (C) A protein non-specifically cross-linking MHC Class II to the TCR, causing massive T cell activation and a cytokine storm
Superantigens are microbial proteins that bypass normal antigen processing. Instead of binding within the MHC peptide-binding groove, they cross-link the conserved outer regions of MHC Class II molecules on antigen-presenting cells directly with specific V-beta regions of T-cell receptors. This interaction non-specifically activates a massive proportion of T cells, triggering an overwhelming, systemic cytokine storm.

NODIA

195. (A) P-2, Q-1, R-2, S-1
Veterinary autoimmune diseases are classified by their primary hypersensitivity mechanisms. Autoimmune hemolytic anemia and pemphigus foliaceus are Type II hypersensitivities, where cytotoxic autoantibodies target surface antigens on erythrocytes and epidermal desmoglein, respectively. In contrast, the vascular lesions of Systemic Lupus Erythematosus and immune-mediated polyarthritis are Type III hypersensitivities driven by soluble immune complexes.

196. (D) Antigen binds B cell → germinal center point mutations → high-affinity selection by follicular dendritic cells → differentiation
Somatic hypermutation and affinity maturation are key processes driving B-cell differentiation. Upon antigen binding, proliferating B cells in the germinal center undergo random point mutations in their immunoglobulin variable genes. FDC-bound native antigens then select the mutated B cells expressing receptors with the highest affinity, which subsequently differentiate into antibody-secresing plasma cells.

197. (B) If a calf fails to nurse within 24 hours of birth, the inevitable lack of absorbed maternal IgG will strictly cause it to instantly develop a permanent genetic defect in its own B cells
Failing to ingest colostrum within twenty-four hours after birth causes failure of passive transfer, leaving the neonate highly vulnerable to infections. However, this lack of passive immunity does not alter the neonate's genome or cause genetic defects. The young animal's B cells remain genetically normal and capable of synthesizing active endogenous antibodies once exposed to antigens.

(D) It is synonymous with complete tetraploidy in domestic animals.

2001

41. Why are autosomal monosomies generally lethal during early embryonic development in mammals?
(A) They cause immediate catastrophic breakdown of the outer cell membrane.
(B) They trigger hyper-transcription of all the remaining autosomes.
(C) They permanently prevent the normal formation of the mitotic spindle.
(D) Unmasked lethal recessive alleles and haploinsufficiency.

2016

42. In contrast to humans, naturally occurring viable autosomal trisomies are extremely rare in domestic animals. What is the primary reason for this?
(A) Severe developmental imbalances lead to early embryonic mortality in animals.
(B) Animal chromosomes do not undergo meiosis.
(C) Domestic animals have highly effective DNA repair mechanisms that correct aneuploidy.
(D) Aneuploid gametes in animals completely fail to fertilize.

2017

43. Which of the following nondisjunction events would produce an XY sperm in a male mammal?
(A) Nondisjunction at Meiosis II
(B) Nondisjunction at Meiosis I
(C) Nondisjunction during spermatogonial mitosis
(D) Crossing over outside the pseudoautosomal region

2004

44. Consider the following statements regarding nondisjunction and aneuploidy:
I. Nondisjunction at Meiosis II produces some normal gametes.
II. Trisomy is a form of euploidy.
III. Monosomy describes a $2n-1$ condition.
IV. Aneuploidy involves the multiplication of complete haploid sets.
Which of the above statements are correct?
(A) I, II and IV
(B) I and III
(C) II, III and IV
(D) I, III and IV

2002

45. What defines a polyploid organism?
(A) It has three or more complete chromosome sets.
(B) It has several missing chromosomes in various homologous pairs.
(C) It contains exactly one additional copy of a single chromosome.
(D) Its academic somatic cells completely lack a distinct nucleus.

2024

46. Triploidy ($3n$) in mammals is most commonly the result of which mechanism?
(A) Meiotic nondisjunction of a single homologous chromosome pair.
(B) The complete structural deletion of a major chromosomal arm.
(C) Dispermy, where two sperm fertilize one ovum.
(D) Centric fusion of two non-homologous acrocentric chromosomes.

2002

47. Which statement accurately reflects the outcome of complete polyploidy (triploidy or tetraploidy) in domesticated mammalian species?
(A) It consistently results in enhanced vigor and significantly larger body size.
(B) It causes complete male sterility while females remain fully fertile.
(C) It directly leads to the formation of genetically distinct chimeras.
(D) It is usually lethal during early embryonic stages.

2002

48. Euploidy refers to variations in chromosome number that:
(A) Involve single individual chromosomes.
(B) Alter the structure but not the number of chromosomes.
(C) Involve whole haploid sets of chromosomes.
(D) Only occur in sex chromosomes.

2016

49. Endoreduplication is a cellular process that can lead to which of the following states?
(A) Autosomal monosomy
(B) Polyploidy
(C) Nullisomy
(D) Chromosomal translocations

2017

50. The artificial induction of polyploidy is a common practice in aquaculture (e.g., oysters

Click Here to Buy book on Flipkart

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

These antigen-antibody complexes activate the classical complement cascade, producing the potent anaphylatoxin C5a. C5a recruits and activates large numbers of neutrophils, which release highly destructive lysosomal enzymes and reactive oxygen species, causing local microvascular damage, thrombosis, and tissue necrosis.

207. (B) Early phase is driven by pre-formed histamine causing vasodilation, while late phase involves lipid mediators and recruited cells
Type I hypersensitivity is divided into distinct physiological phases. The early-phase response occurs within minutes, driven by pre-formed histamine released from degranulating mast cells, which causes immediate vasodilation and bronchoconstriction. In contrast, the late-phase response occurs hours later, characterized by the synthesis of lipid mediators and recruitment of eosinophils and inflammatory neutrophils.

208. (D) It strictly eliminates perfectly functioning, highly reactive pathogen-specific lymphocytes to save energy
Central tolerance is an essential safety mechanism that occurs in primary lymphoid organs. This process selectively eliminates immature lymphocytes that react with high affinity to self-antigens, predominantly through negative selection and apoptosis. Central tolerance does not eliminate safe, functional, pathogen-specific lymphocytes, as these are vital for maintaining host protection against foreign infections.

209. (A) Autoantibodies block and degrade acetylcholine receptors at the neuromuscular junction, causing skeletal muscle weakness
Myasthenia Gravis is a classic autoantibody-mediated disease of the neuromuscular junction. Affected animals produce specific autoantibodies targeting nicotinic acetylcholine receptors on the postsynaptic membrane. This binding blocks receptor function and accelerates their degradation. Consequently, neuromuscular transmission is severely impaired, resulting in progressive skeletal muscle weakness and exercise intolerance in affected domestic dogs.

210. (C) An antibody or TCR elicited by one antigen binds to a different, structurally similar antigen
Immunological cross-reactivity is a fundamental concept where an adaptive immune response targets multiple antigens. It occurs when an antibody or T-cell receptor raised against a specific epitope also binds to a different antigen. This binding is driven by shared structural motifs or sequence homologies, and it is the mechanism underlying molecular mimicry.

****

Downloaded from www.pdf.tube

CHAPTER 05
Genetically Determined Diseases

NODIA

2015
1. Which of the following mutations results in a premature termination codon within the transcribed mRNA?
(A) Missense mutation
(B) Nonsense mutation
(C) Silent mutation
(D) Frameshift mutation

2005
2. The central dogma of molecular biology describes the directional flow of genetic information. Which of the following pathways represents the standard flow according to this principle?
(A) RNA to DNA to Protein
(B) Protein to RNA to DNA
(C) RNA to Protein to DNA
(D) DNA to RNA to Protein

2013
3. What is the primary molecular consequence of an insertion or deletion of a single nucleotide within a coding region?
(A) A shift in the reading frame of the ribosome
(B) An exact substitution of a single amino acid
(C) Immediate degradation of the DNA sequence
(D) Failure of the RNA polymerase to bind the promoter

2015
4. Which of the following statements INCORRECTLY describes a point mutation?
(A) It involves the substitution of a single base pair.
(B) It can result in a missense mutation within exons.
(C) It always alters the total polypeptide length.
(D) It can be classified as a transition or a transversion.

2006
5. A genetic alteration converts a codon specifying an amino acid into a codon specifying a different amino acid. What type of mutation has occurred?
(A) Missense mutation
(B) Nonsense mutation
(C) Frameshift mutation
(D) Synonymous mutation

2018
6. Which phenomenon best explains how multiple different codons can translate into the same amino acid, thereby masking some point mutations?
(A) Wobble base pairing rules
(B) Universal genetic code system
(C) Alternative mRNA splicing
(D) Genetic code degeneracy

2005
7. What is the defining characteristic of a transversion point mutation?
(A) A purine is replaced by another purine base.
(B) A purine is replaced by a pyrimidine base.
(C) A pyrimidine is replaced by another pyrimidine.
(D) An entire codon is inverted in genetic sequence.

2018
8. Match the mutation types in List I with their primary consequences in List II.
List I: Mutation Types
P. Point transition
Q. Frameshift
R. Nonsense
S. Silent
List II: Primary Consequences
1. Altered reading frame
2. Premature stop codon
3. No change in amino acid
4. Purine to purine change
(A) P-1, Q-4, R-3, S-2
(B) P-4, Q-2, R-1, S-3
(C) P-4, Q-1, R-2, S-3
(D) P-3, Q-1, R-4, S-2

2022
9. Exposure to ethidium bromide primarily induces which specific class of genetic mutations during replication?
(A) Nonsense-type mutations
(B) Missense-type mutations
(C) Silent-type mutations
(D) Frameshift mutations

2019

10. In an autosomal dominant inherited disease, what is the expected phenotypic outcome for heterozygous individuals?
(A) They will express the disease phenotype.
(B) They will appear clinically normal but act as carriers.
(C) They will express a mild, intermediate form of the disease.
(D) They will only express the disease if exposed to specific environmental triggers.

2013

11. Which characteristic is a hallmark of X-linked recessive inheritance in mammals?
(A) Affected males and females are equally distributed in a population.
(B) Affected females inherit the allele from both parents.
(C) Affected males always transmit the trait to all of their sons.
(D) The mutated trait can be passed directly from father to son.

2018

12. If two heterozygous carriers of an autosomal recessive lethal trait are mated, what is the expected genotypic ratio among the surviving offspring?
(A) 1 homozygous normal : 2 heterozygous carriers
(B) 1 homozygous normal : 3 heterozygous carriers
(C) 2 homozygous normal : 1 affected
(D) 1 homozygous normal : 1 affected

2001

13. Which inheritance pattern typically causes a disease to skip generations, appearing only when two carriers reproduce?
(A) Autosomal recessive
(B) Autosomal dominant
(C) Mitochondrial inheritance
(D) X-linked dominant

2007

14. An inherited disorder affects every generation of a pedigree, and affected males transmit the disease to all their daughters but none of their sons. What is the most likely mode of inheritance?
(A) Autosomal dominant
(B) X-linked recessive
(C) X-linked dominant
(D) Y-linked

2015

15. The concept of a proband in medical genetics refers to:
(A) The ancestral founder of a specific genetic mutation in a population.
(B) A clinically asymptomatic carrier who shows no physical symptoms.
(C) A heterozygous individual who possesses a single lethal allele.
(D) The first affected individual evaluated in the family.

2008

16. In the context of autosomal recessive disorders, why does inbreeding increase the incidence of affected individuals?
(A) It increases the overall mutation rate in the population.
(B) It promotes chromosomal nondisjunction.
(C) It increases the probability of homozygosity by descent.
(D) It introduces new lethal alleles from other populations.

2004

17. Given the following Assertion (A) and **Reason (R):**
Assertion (A): Males are more frequently affected by X-linked recessive disorders than females.
Reason (R): Males are hemizygous for the X chromosome, so a single recessive allele results in phenotypic expression.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2023

18. Which statement is an exception to classic Mendelian inheritance patterns?
(A) Segregation of alleles occurs during gametogenesis.
(B) Independent assortment of genes located on different chromosomes.
(C) Recessive alleles are masked by dominant alleles in heterozygotes.
(D) Genomic imprinting based on parental origin.

2019

19. What term describes the percentage of individuals with a specific genotype who exhibit the corresponding phenotype?
(A) Penetrance

Click Here to Buy book on Amazon

(B) Expressivity
(C) Pleiotropy
(D) Epistasis

2024

20. Variable expressivity in a genetic disorder implies that:
(A) Not all individuals carrying the mutant genotype display the phenotype.
(B) A single genetic locus influences multiple, seemingly unrelated traits.
(C) Individuals with the same genotype show varying clinical severity.
(D) Environmental and external factors entirely mask the genetic defect.

2021

21. Marfan syndrome in humans and analogous connective tissue disorders in animals cause defects in the skeleton, eyes, and cardiovascular system. This is a classic example of:
(A) Pleiotropy
(B) Incomplete dominance
(C) Variable expressivity
(D) Polygenic inheritance

2012

22. Which of the following factors does NOT influence the penetrance of a genetic trait?
(A) Epigenetic modifications
(B) Modifier genes
(C) Environmental influences
(D) Independent assortment

2011

23. A livestock breeder observes that a particular dominant gene for horn presence is inherited, but only 80% of the animals carrying the gene actually develop horns. This illustrates:
(A) Codominant gene expression
(B) Incomplete penetrance
(C) Pleiotropic gene action
(D) Genomic imprinting pattern

2009

24. Which scenario perfectly illustrates the concept of pleiotropy in domestic animals?
(A) The genetic interaction of two distinct loci determining coat color in horses.
(B) Seasonal environmental temperatures dictating point coloration in Siamese cats.
(C) The Merle gene causing coat patterning and deafness in dogs.
(D) Variable phenotypic heights in a herd of cattle highly influenced by nutrition.

2010

25. Age-dependent penetrance is a recognized phenomenon in several genetic diseases. What does this mean?
(A) The severity of the disease decreases as the animal ages.
(B) The disease allele is only transcribed during fetal development.
(C) The mutation rate increases proportionally with parental age.
(D) Clinical signs of the disorder manifest later in life.

2017

26. Consider the following statements regarding genetic expression phenomena:
I. Penetrance refers to the varying severity of a phenotype.
II. Pleiotropy occurs when one gene affects multiple phenotypic traits.
III. Incomplete penetrance means some individuals with the genotype show no phenotype.
Which of the above statements are correct?
(A) II and III
(B) I and II
(C) I
(D) I and III

2014

27. Which chemical agent is commonly used in karyotyping to arrest cells in metaphase by inhibiting spindle fiber formation?
(A) Phytohemagglutinin
(B) Colchicine
(C) Giemsa
(D) Ethidium bromide

2004

28. In cytogenetics, the standard technique used to produce alternating dark and light bands along the length of chromosomes is known as:
(A) C-banding
(B) G-banding
(C) R-banding
(D) Q-banding

2002

29. What does the dark band in a standard G-banded chromosome primarily represent?
(A) AT-rich, late replicating heterochromatin
(B) GC-rich, transcriptionally active regions
(C) AT-rich, early replicating regions
(D) Telomeric and centromeric sequences strictly

NODIA

2024

30. Which term specifically defines the systematic arrangement of homologous chromosomes in descending order of size?
(A) Idiogram
(B) Pedigree chart
(C) Karyotype
(D) Cladogram tree

2002

31. According to standard cytogenetic nomenclature, what does the designation $2n = 60, XX$ signify in cattle?
(A) A normal male karyotype with 60 chromosomes
(B) An aneuploid female missing a pair of chromosomes
(C) A polyploid cell line with 60 pairs of chromosomes
(D) A normal female karyotype with 60 chromosomes

2012

32. Which banding technique is specifically utilized to stain constitutive heterochromatin, primarily located at the centromeres?
(A) G-banding
(B) NOR-banding
(C) C-banding
(D) R-banding

2008

33. The short arm of a chromosome is denoted in cytogenetic nomenclature by which letter?
(A) q
(B) p
(C) s
(D) t

2017

34. In evaluating an ideogram, how are the bands numbered on a chromosome?
(A) From the physical telomere inward towards the centromere
(B) From the long arm to the short arm in consecutive order
(C) Randomly based on the relative intensity of the stain
(D) From the centromere outward to the telomeres

2010

35. Select the correct order of the standard procedures involved in preparing a metaphase chromosome spread for karyotyping:
(A) Cell culture → Colchicine treatment → Hypotonic treatment → Fixation → Staining
(B) Cell culture → Hypotonic treatment → Colchicine treatment → Staining → Fixation
(C) Colchicine treatment → Cell culture → Fixation → Hypotonic treatment → Staining
(D) Cell culture → Fixation → Hypotonic treatment → Colchicine treatment → Staining

2001

36. Which biological process is the primary cause of numerical chromosomal aneuploidy?
(A) Crossing over during prophase I
(B) Unequal sister chromatid exchange
(C) Nondisjunction during meiosis or mitosis
(D) DNA polymerase slippage during replication

2018

37. What chromosomal state is denoted by the formula $2n - 1$?
(A) Monosomy
(B) Nullisomy
(C) Trisomy
(D) Tetrasomy

2025

38. If nondisjunction of a single chromosome pair occurs during Meiosis I, what will be the chromosomal complement of the resulting four gametes?
(A) Two will be normal n, one $n+1$, and one $n-1$
(B) All four will be $n+1$
(C) All four will be $n-1$
(D) Two will be $n+1$ and two will be $n-1$

2002

39. Nullisomy is a condition best defined by which of the following expressions?
(A) Loss of one chromosome from a homologous pair
(B) Loss of both chromosomes of a homologous pair
(C) Addition of a single extra chromosome
(D) Addition of an entire haploid set of chromosomes

2008

40. Which of the following statements about tetrasomy is strictly correct?
(A) It involves the loss of four separate and distinct chromosomes.
(B) It results in a total somatic cell chromosome number of $4n$.
(C) It involves the gain of a homologous chromosome pair.

Downloaded from www.pdf.tube

198. (C) The endotheliochorial placenta allows 5-10% pre-partum IgG transfer, whereas the epitheliochorial blocks all transfer
Placentation determines the degree of prenatal immunoglobulin transfer in domestic species. Cat and dog endotheliochorial placentas allow minor maternal-fetal transfer, delivering five to ten percent of neonatal IgG in utero. Conversely, horse and pig epitheliochorial placentas are entirely impermeable to large immunoglobulins, rendering the neonate completely agammaglobulinemic at birth and reliant on colostrum.

199. (D) Th1 cells secrete IFN-gamma, promoting classical macrophage activation and inhibiting Th2 proliferation
The balance between Th1 and Th2 adaptive immune responses is regulated by specific cytokine networks. Activated Th1 cells secrete high concentrations of interferon-gamma. Interferon-gamma promotes classical macrophage activation and cell-mediated immunity while directly inhibiting the proliferation of Th2 helper T cells, thereby suppressing the competing humoral and allergic response pathway in the host.

200. (A) IgD is the primary structural component of the classical complement Membrane Attack Complex
Immunoglobulin isotypes possess distinct structural designs and physiological roles. IgA is a secretory dimer protecting mucosal surfaces, IgE is a cell-bound monomer mediating allergic responses, and IgM is a large pentamer dominating primary humoral responses. IgD is a membrane-bound receptor on naïve B cells; it is not a component of the complement Membrane Attack Complex.

201. (B) Re-exposure → Th1 recognition → IFN-gamma secretion → macrophage recruitment/ activation → induration at 48-72 hours
A cell-mediated Type IV delayed-type hypersensitivity reaction follows a precise timeline. Upon re-exposure, sensitized Th1 cells recognize the target antigen and secrete interferon-gamma. This cytokine recruits and activates large numbers of macrophages at the site. Because this cellular infiltration requires protein synthesis and migration, visible induration only peaks forty-eight to seventy-two hours later.

202. (B) Profound immunodeficiency with absent mature T cells, preventing cell-mediated and T-dependent antibody responses
The thymus is the essential primary lymphoid organ responsible for T-lymphocyte maturation and selection. In thymic aplasia, mature functional T cells are entirely absent from circulation. Because helper T cells are required to coordinate cell-mediated defenses and stimulate B-cell class switching, affected animals exhibit profound deficiencies in both cellular and humoral immunity.

203. (A) Terminal Pathway : Initiated strictly by the direct binding of free histamine to C9
The complement system pathways are initiated by distinct biomolecules. The classical pathway is triggered by IgG or IgM complexes, the alternative pathway by spontaneous hydrolysis, and the lectin pathway by carbohydrate binding. The terminal pathway is initiated when C5 convertase cleaves C5 to form C5b, not by the direct binding of free histamine to C9.

204. (C) Enterocytes capable of pinocytosing immunoglobulins are replaced by mature cells, ending passive transfer
Gut closure is a major physiological event in neonatal development. Over the first twenty-four hours of life, specialized enterocytes capable of non-specific pinocytosis are rapidly replaced by mature, impermeable epithelial cells. This transition ends the absorption of intact colostral immunoglobulins into the bloodstream, limiting the window for acquiring passive systemic immunity in ruminants.

205. (D) P-3, Q-4, R-1, S-2
Adaptive immunity depends on structurally distinct chemical components. Antigens are molecules recognized by lymphocytes. Antibodies are soluble glycoprotein complexes secreted by plasma cells. Adjuvants are compounds added to vaccines to enhance host immunogenicity without serving as target antigens, and haptens are small non-immunogenic molecules requiring carrier conjugation to elicit an immune response in vivo.

NODIA

206. (C) Complexes activate complement, recruiting neutrophils via C5a that release destructive lysosomal enzymes and oxygen radicals
Localized deposition of immune complexes drives severe tissue necrosis in the Arthus reaction.

and salmon). What is the primary economic advantage of induced triploidy in these species?
(A) Significantly increased disease resistance against various viral pathogens.
(B) Enhanced reproductive capacity and higher overall egg viability.
(C) Rapid development of highly diverse and attractive coloration patterns.
(D) Sterility, diverting energy to somatic growth.

2023
51. Which of the following conditions represents a diploid animal that has retained an unreduced polar body during oogenesis, leading to subsequent fertilization?
(A) Triploidy
(B) Chromosomal nullisomy
(C) Autosomal trisomy
(D) Cellular haploidy

2005
52. Why does triploidy strictly result in sterility even if an organism survives to adulthood?
(A) Triploid somatic and germ cells undergo rapid, widespread apoptosis during puberty.
(B) Having three complete sets of genes severely represses gonadotropin-releasing hormone.
(C) Odd chromosome sets cannot pair properly in meiosis, yielding unbalanced gametes.
(D) All essential sex chromosomes are completely missing or deactivated in triploids.

2024
53. Given the following Assertion (A) and **Reason (R):**
Assertion (A): Triploidy is frequently observed in live-born poultry, leading to phenotypic abnormalities.
Reason (R): Avian species tolerate chromosomal imbalances much better than mammals due to microchromosomes.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2002
54. What type of chromosomal structural aberration is characterized by the loss of an internal chromosomal segment?
(A) Terminal deletion
(B) Pericentric chromosomal inversion
(C) Tandem duplication
(D) Interstitial deletion

2011
55. A segment of a chromosome breaks off, rotates 180 degrees, and reattaches. If the inverted segment does NOT include the centromere, it is termed:
(A) Pericentric inversion
(B) Paracentric inversion
(C) Reciprocal translocation
(D) Isochromosome formation

2002
56. Which consequence is typical when crossing over occurs within the inversion loop of a paracentric inversion heterozygote?
(A) Complete formation of viable normal and structurally inverted gametes only.
(B) Immediate physical creation of a stable, functional ring chromosome.
(C) Production of dicentric and acentric chromatids.
(D) Complete and spontaneous nondisjunction of the entire homologous bivalent.

2011
57. Duplications in a genome often play a crucial evolutionary role because they:
(A) Suppress meiotic recombination events entirely across the homologous chromosomes.
(B) Actively delete harmful recessive mutations through natural selection pathways.
(C) Cause immediate and complete reproductive isolation within the population.
(D) Provide new genetic material for the evolution of gene families.

2019
58. An organism heterozygous for a large chromosomal deletion is most likely to exhibit which of the following genetic phenomena?
(A) Pseudodominance
(B) Overdominance
(C) Complete penetrance
(D) Codominance

2015
59. What visual feature differentiates a pericentric inversion from a paracentric inversion when examining a standard karyotype?
(A) Paracentric inversions always change the arm ratio and centromeric index.
(B) Pericentric inversions may alter the chromosome arm ratio.
(C) Pericentric inversions create a distinct acentric fragment visible in metaphase.

Click Here to Buy book on Flipkart

(D) Paracentric inversions lead directly to the formation of a Robertsonian fusion.

2017

60. Cri-du-chat syndrome in humans is analogous to certain animal conditions caused by which specific chromosomal aberration?
(A) A large pericentric chromosomal inversion
(B) A partial short arm deletion
(C) A tandem chromosomal duplication
(D) A balanced Robertsonian translocation

2008

61. Which of the following structural aberrations requires three distinct chromosome breaks to occur?
(A) Interstitial deletion
(B) Paracentric inversion
(C) Chromosomal insertion
(D) Reciprocal translocation

2022

62. Match the structural aberrations in List I with their defining characteristics in List II.
List I: Structural Aberrations
P. Paracentric inversion
Q. Pericentric inversion
R. Deletion
S. Duplication
List II: Defining Characteristics
1. Centromere is included in the inverted region
2. Leads to loss of genetic material
3. Centromere is outside the inverted region
4. Results in a repeated segment
(A) P-1, Q-3, R-2, S-4
(B) P-3, Q-1, R-4, S-2
(C) P-3, Q-1, R-2, S-4
(D) P-1, Q-3, R-4, S-2

2011

63. What is the fundamental mechanism of a reciprocal translocation?
(A) Exchange of segments between homologous chromosomes during meiosis
(B) Fusion of two acrocentric chromosomes at their centromeres
(C) Reversal of a segment within a single chromosome
(D) Exchange of segments between two non-homologous chromosomes

2012

64. The most famous chromosomal abnormality in cattle, the 1/29 translocation, involves which type of structural rearrangement?
(A) Reciprocal chromosomal translocation
(B) Pericentric chromosomal inversion
(C) Robertsonian translocation
(D) Interstitial chromosomal deletion

2013

65. What is the primary phenotypic consequence of the 1/29 translocation in heterozygous carrier cattle?
(A) Lethal achondroplasia and systemic skeletal dwarfism
(B) Reduced fertility from embryonic mortality
(C) Severe immunodeficiency making animals prone to infection
(D) Abnormal development of intersex physical characteristics

2001

66. A Robertsonian translocation typically involves the fusion of which types of chromosomes?
(A) Two metacentric chromosomes
(B) One metacentric and one acrocentric chromosome
(C) Any two non-homologous autosomes and sex chromosomes
(D) Two acrocentric or telocentric chromosomes

2010

67. How does a Robertsonian translocation structurally alter the karyotype of a balanced carrier?
(A) It reduces the total chromosome number by one.
(B) It increases the basic chromosome number by one through duplication.
(C) It results in a massive, catastrophic deletion of euchromatin.
(D) It replaces the Y chromosome with an autosome during replication.

2016

68. During meiosis in an individual heterozygous for a reciprocal translocation, how do the chromosomes typically align?
(A) They form a bivalent loop structure.
(B) They immediately degrade due to structural tension.
(C) They form a cruciform (cross-shaped) quadrivalent.
(D) They fail to pair, resulting in univalents.

2004

69. In a reciprocal translocation heterozygote, which mode of segregation during anaphase I results in balanced, viable gametes?
(A) Alternate segregation
(B) Adjacent-1 segregation

NODIA

(C) Adjacent-2 segregation
(D) Nondisjunction

2003

70. The 1/29 translocation is most prevalent in which specific breed of cattle, where it was first discovered?
(A) Holstein-Friesian dairy
(B) Hereford beef breed
(C) Aberdeen Angus breed
(D) Swedish Red and White

2003

71. Consider the following statements regarding the 1/29 Robertsonian translocation:
I. It involves the fusion of chromosomes 1 and 29.
II. Heterozygous carriers have 59 chromosomes instead of 60.
III. Homozygous carriers are always prenatally lethal.
IV. It primarily causes reduced fertility in carrier bulls and cows.
V. It is a form of reciprocal translocation.
Which of the above statements are correct?
(A) I, II and V
(B) I, II and IV
(C) II, III and V
(D) I, IV and V

2013

72. The animal equivalent of human Turner's syndrome exhibits which sex chromosome karyotype?
(A) XXY
(B) XYY
(C) XO
(D) XXX

2014

73. What is the primary clinical manifestation of the XO chromosomal anomaly in domestic animals, such as mares?
(A) Aggressive male-like behavior
(B) Severe skeletal dwarfism defects
(C) Increased hyperfertility traits
(D) Gonadal dysgenesis

2008

74. Which karyotype is analogous to Klinefelter's syndrome in male mammals?
(A) XXY
(B) XYY
(C) XO
(D) XX/XY

2003

75. Male tortoiseshell or calico cats are extremely rare. When they do occur, they typically possess which chromosomal anomaly?
(A) XO
(B) XXY
(C) XYY
(D) Complete androgen insensitivity

2019

76. Why are individuals with an XXY karyotype generally sterile?
(A) Lack of the SRY gene on the short arm of the Y chromosome.
(B) Testicular hypoplasia and lack of sperm.
(C) Complete, absolute absence of male reproductive organs.
(D) Inability of Leydig cells to produce testosterone entirely.

2012

77. Which sex chromosome aneuploidy in animals is primarily associated with females exhibiting small stature, lack of estrus cycles, and hypoplastic ovaries?
(A) Turner's-like syndrome (XO)
(B) XXX syndrome
(C) Klinefelter's-like syndrome (XXY)
(D) Freemartinism

NODIA

2016

78. In male mammals bearing the XXY karyotype, the presence of the Y chromosome ensures the development of:
(A) Fully functional, active ovaries
(B) A functioning, complete uterus
(C) Phenotypically male anatomy.
(D) A fertile and active sperm line

2017

79. The XYY syndrome, sometimes recorded in domestic animals, typically results from nondisjunction during which specific meiotic stage?
(A) Paternal Meiosis I
(B) Maternal Meiosis I
(C) Maternal Meiosis II division
(D) Paternal Meiosis II

2025

80. Arrange the following steps sequentially in the developmental pathogenesis of XXY syndrome in a mammal:
1. Meiotic nondisjunction in a parent
2. Testicular hypoplasia during maturation
3. Fertilization producing an XXY zygote

(A) Myostatin
(B) Uridine Monophosphate Synthase
(C) Argininosuccinate synthetase
(D) Beta-2 integrin (CD18)

2006

136. What is the primary clinical manifestation of a calf homozygous for the BLAD mutation?
(A) Recurrent, severe bacterial infections and early death
(B) Uncontrolled hemorrhage and generalized systemic bleeding
(C) Rapid skeletal muscle hypertrophy and physical overgrowth
(D) Complete skeletal muscle paralysis and motor dysfunction

2015

137. Which specific molecular anomaly prevents neutrophil migration in calves with BLAD?
(A) A structural deficiency causing a total lack of cilia in mature neutrophils
(B) A CD18 defect preventing neutrophil adhesion to blood vessel walls
(C) A complete bone marrow failure to produce or differentiate viable neutrophils
(D) A regulatory mutation triggering premature apoptosis within the bone marrow

2022

138. Complex Vertebral Malformation (CVM) in Holstein cattle is a lethal genetic defect inherited in an autosomal recessive manner. It is caused by a missense mutation in which gene?
(A) ITGB2
(B) SLC35A3
(C) UMPS
(D) ASS1

2008

139. The mutation causing CVM (Complex Vertebral Malformation) impairs which cellular process?
(A) UDP-N-acetylglucosamine transport into the Golgi
(B) DNA replication during mitotic division of chondrocytes
(C) Translation of structural type-II collagen proteins
(D) Meiotic spindle formation in germline precursor cells

2018

140. Calves born with CVM commonly present with which set of physical anomalies?
(A) Severe generalized muscle weakness and bilateral congenital blindness
(B) Complete absence of thoracic limbs and severe congenital hydrocephalus
(C) Shortened neck, scoliosis, and bilateral arthrogryposis
(D) Bilateral polycystic kidney disease and progressive hepatic failure

2011

141. How did the BLAD and CVM mutations become so widespread within the global Holstein-Friesian cattle population?
(A) Spontaneous independent mutations occurring across multiple herds
(B) Heterozygote resistance to clinical status... wait, preserve text exactly: "Heterozygote resistance to clinical mastitis in dairy systems"
(B) Heterozygote resistance to clinical mastitis in dairy systems
(C) Uncontrolled crossbreeding with various beef cattle populations
(D) Extensive use of heavily favored AI carrier sires

2007

142. If a carrier bull for CVM is mated with a carrier cow, what is the probability that a resulting liveborn calf is a carrier? (Assume homozygous recessives are aborted prenatally or die immediately).
(A) 25%
(B) 50%
(C) 66.7%
(D) 100%

2017

143. These questions consist of two statements each, printed as Assertion and Reason. Answer the questions selecting the appropriate option.
Assertion (A): DUMPS (Deficiency of Uridine Monophosphate Synthase) is maintained at a low frequency in dairy populations primarily because the homozygous condition causes embryonic death.
Reason (R): Homozygous embryos fail to synthesize pyrimidine nucleotides de novo, leading to lethality around day 40 of gestation.
(A) Both (A) and (R) are true and (R) is the correct explanation of (A).
(B) Both (A) and (R) are true but (R) is not the correct explanation of (A).
(C) (A) is true but (R) is false.
(D) (A) is false but (R) is true.

Downloaded from www.pdf.tube

a chromosome from a single parent, which can result from a "trisomy rescue" event?
(A) Genomic imprinting
(B) Uniparental disomy
(C) Gonadal mosaicism
(D) Biparental inheritance

2022
91. When an autosomal trisomy is identified in a spontaneously aborted bovine fetus, the most prevalent mechanism leading to this karyotype is:
(A) Fertilization by two sperm cells
(B) Maternal meiotic nondisjunction
(C) Mitotic nondisjunction during the blastocyst stage
(D) Robertsonian centric fusion

2009
92. Trisomy 18 (analogous to Edwards syndrome) in mammals is typically characterized by which set of pathological manifestations?
(A) Rapid, completely unchecked somatic growth and development
(B) Severe craniofacial, cardiac, and skeletal defects
(C) Exclusive developmental malformation of the reproductive tract
(D) No visible physical manifestations, presenting only sterility

2007
93. A livestock pathology report indicates a fetus was aborted due to Trisomy 16. The basic chromosomal condition of the somatic cells of this fetus was:
(A) $2n+1$
(B) $2n-1$
(C) $3n$
(D) $2n+2$

2019
94. Among the rare reports of live-born animals with autosomal trisomies, the most notable clinical finding across species is:
(A) Superior athletic performance and speed
(B) Exceptionally prolonged lifespan expectancy
(C) Multiple congenital malformations
(D) Complete resistance to common viral infections

2002
95. What role does advanced maternal age play in the occurrence of autosomal trisomies in domestic animals?
(A) It significantly decreases the overall likelihood of meiotic nondisjunction events.
(B) It causes paternal sperm to undergo mitotic nondisjunction immediately upon fertilization.
(C) It has absolutely no direct correlation with chromosomal aneuploidy in domestic species.
(D) It increases nondisjunction due to prolonged oocyte arrest in dictyotene.

2024
96. The pathological manifestations of autosomal trisomies are primarily caused by:
(A) The complete developmental absence of multiple crucial homeobox genes.
(B) The physical crowding of abnormal chromosomes inside the active cell nucleus.
(C) Gene dosage effects from triplicated chromosome overexpression.
(D) Severe generalized failure of random X-chromosome inactivation in embryos.

2024
97. The phenomenon where a trisomic cell line loses the extra chromosome during early embryogenesis, potentially leading to uniparental disomy, is termed:
(A) Endoreduplication
(B) Trisomy rescue
(C) Centric fusion
(D) Heterochromatinization

2018
98. Given the following Assertion (A) and **Reason (R):**
Assertion (A): Mosaicism can result in an individual having both normal $(2n)$ and aneuploid $(2n+1)$ cell lines.
Reason (R): Nondisjunction occurring exclusively during post-zygotic mitotic divisions generates distinct cellular populations.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

NODIA

2006
99. How is genetic mosaicism fundamentally defined?
(A) The presence of two or more genetically distinct cell lines originating from a single zygote.
(B) The presence of two or more genetically distinct cell lines originating from different zygotes.

(C) A temporary fluctuation in gene expression due to environmental factors.
(D) The fusion of twin embryos in utero.

2023
100. Which of the following events is the most common cause of chromosomal mosaicism?
(A) Meiotic nondisjunction events during oogenesis in the mother.
(B) Meiotic nondisjunction events during spermatogenesis in the father.
(C) Mitotic nondisjunction in early cleavage.
(D) Independent assortment of non-homologous chromosomes in gametes.

2022
101. Anaphase lag is a mechanism that typically results in the formation of:
(A) A monosomic cell line
(B) A trisomic cell line
(C) A triploid cell line
(D) A tetrasomic cell line

2018
102. If a somatic mutation occurs in a skin cell lineage during early fetal development of an animal, the resulting phenotype will likely present as:
(A) The entire animal's body uniformly exhibiting the mutated phenotype.
(B) The somatic mutation being isolated strictly to the developing germ cells.
(C) Immediate and complete fetal death within the maternal womb.
(D) A localized patch of skin showing the mutated phenotype.

2013
103. Which clinical outcome is generally associated with an individual who is a mosaic for an autosomal trisomy (e.g., 46,XX / 47,XX,+21) compared to a fully trisomic individual?
(A) A more severe phenotype than full trisomy
(B) A milder phenotypic expression of the syndrome
(C) A completely normal phenotype without exceptions
(D) Complete sterility without any somatic abnormalities

2010
104. Germline mosaicism implies that a mutation is present:
(A) Exclusively in the somatic tissues such as skin, muscle, and liver.
(B) Uniformly in every single somatic and reproductive cell of the body.
(C) In a proportion of gametes but not somatic cells.
(D) Only in the mitochondrial DNA of maternal-line somatic tissues.

2023
105. Why is germline mosaicism particularly important in genetic counseling and breeding programs?
(A) It quickly renders the heterozygous parent completely sterile.
(B) It causes the parent to show severe progressive clinical signs later in life.
(C) It severely alters the karyotype of the parent's somatic blood cells.
(D) Normal parents can repeatedly produce affected offspring.

2018
106. A post-zygotic mutation in the early blastocyst stage will affect:
(A) All somatic and germline cells of the adult organism
(B) A subset of somatic tissues in the adult organism
(C) Only the extraembryonic membranes and placental tissues
(D) Exclusively the central and peripheral nervous system

2019
107. Match the genetic phenomenon listed in List I with its defining mechanism in List II:
List I: (Genetic phenomenon)
P. X-inactivation
Q. Somatic Mosaicism
R. Germline Mosaicism
S. Anaphase lag
List II: (Defining mechanism)
1. Mutation isolated to gamete precursors
2. Post-zygotic loss of a chromosome
3. Epigenetic silencing of a chromosome
4. Post-zygotic mutation in body tissues
(A) P-3, Q-4, R-1, S-2
(B) P-3, Q-1, R-4, S-2
(C) P-2, Q-4, R-1, S-3
(D) P-1, Q-2, R-3, S-4

2007
108. The Lyon hypothesis describes which crucial genetic mechanism in female mammals?
(A) Preferential epigenetic silencing of the maternal X chromosome
(B) Transcriptional inactivation of the Y chromosome in males

Click Here to Buy book on Amazon

(C) Random epigenetic silencing of one X chromosome in females
(D) Selective mutational silencing of the pseudoautosomal regions

2007
109. What is the primary biological purpose of X-chromosome inactivation?
(A) To prevent deleterious recessive mutations from expressing in female somatic tissues
(B) To induce random somatic mosaicism across all functional mammalian organ systems
(C) To regulate hormone levels and control the onset of puberty in young females
(D) To equalize the dosage of X-linked gene expression between males and females

2012
110. The inactive X chromosome in a female mammalian cell can be visualised under a microscope as a dense structure known as:
(A) A nucleolus
(B) A kinetochore
(C) A Barr body
(D) A synaptonemal complex

2016
111. Which specific RNA molecule is responsible for coating and initiating the inactivation of the X chromosome?
(A) mRNA
(B) XIST ncRNA
(C) rRNA
(D) tRNA

2008
112. The tortoiseshell coat pattern in female cats is a classic example of:
(A) Codominance between autosomal pigmentation alleles
(B) Incomplete dominance of autosomal coat color genes
(C) Epistatic interactions of multiple modifier loci
(D) Mosaicism driven by X-chromosome inactivation

2009
113. Why are typical tortoiseshell cats almost exclusively female?
(A) The orange/black gene is X-linked, requiring two X chromosomes for mosaicism.
(B) The genes regulating black and orange coat colors are embryonically lethal in males.
(C) Hemizygous males completely lack the specialized hair follicles to express orange.
(D) High levels of circulating testosterone actively suppress the orange allele expression.

NODIA

2015
114. If a male cat is found to have a genuine tortoiseshell coat pattern, what is his most likely sex chromosome karyotype?
(A) XY
(B) XYY
(C) XXY
(D) XO

2014
115. Once an X chromosome is randomly inactivated in an embryonic cell, what happens during subsequent mitotic divisions of that cell?
(A) The same X chromosome remains inactive in all descendant cells.
(B) The inactive X chromosome becomes reactivated.
(C) The choice of which X is inactive is randomized again at each division.
(D) Both X chromosomes become completely active.

2004
116. Consider the following statements regarding chimerism:
I. A chimera derives from a single fertilization event.
II. Tetragametic chimeras form from the fusion of two separate zygotes.
III. Blood chimerism in cattle is entirely dependent on meiotic nondisjunction.
IV. Freemartinism involves hematopoietic chimerism.
Which of the above statements are INCORRECT?
(A) II and IV
(B) I and II
(C) III and IV
(D) I and III

2008
117. What distinguishes a true genetic chimera from a genetic mosaic?
(A) Mosaics have cells with different chromosomes; a chimera has structural anomalies.
(B) Mosaics originate from one zygote; chimeras from two or more distinct zygotes.
(C) Mosaics occur exclusively in females; chimeras occur exclusively in developing males.
(D) Mosaics exhibit localized tissue defects; chimeras exhibit widespread systemic

4. Activation of SRY gene driving testis formation

(A) 1, 4, 3, 2
(B) 3, 1, 4, 2
(C) 1, 3, 4, 2
(D) 1, 3, 2, 4

2022

81. The SRY gene is critical for mammalian sex determination. Where is it typically located in a normal male karyotype?
(A) On the long arm of the X chromosome
(B) On the short arm of the Y chromosome
(C) On an autosome
(D) In the pseudoautosomal region of X and Y

2008

82. What is the primary function of the SRY gene product?
(A) It actively produces testosterone hormone in Leydig cells.
(B) It degrades the female Müllerian ducts directly via protein synthesis.
(C) It promotes the development of the male Wolffian ducts directly.
(D) It initiates indifferent gonad development into a testis.

2007

83. Polled Intersex Syndrome (PIS) in dairy goats is an anomaly intricately linked to which specific trait?
(A) Polled trait (hornless)
(B) Inherited ear shape characteristics
(C) Specific coat color variations
(D) Congenital dwarfism mutations

2017

84. In goats exhibiting Polled Intersex Syndrome (PIS), what is the typical chromosomal sex and corresponding phenotype of the affected intersex individuals?
(A) XY karyotype with complete female phenotype
(B) XO karyotype with distinct intersex phenotype
(C) XX karyotype with intersex phenotype
(D) XXY karyotype with normal female-like phenotype

2012

85. Polled Intersex Syndrome (PIS) in goats is caused by a large genomic deletion that impacts the regulatory elements of which key sex-determining genes?
(A) FOXL2 and PISRT1
(B) SRY and AMH pathways
(C) SOX9 and DMRT1 systems
(D) WT1 and SF1 factors

2001

86. XX sex reversal in breeds like the Cocker Spaniel is a condition where individuals are genetically female (XX) but develop male gonads. In dogs, this condition is uniquely characterized by:
(A) The presence of a translocated SRY gene on the X chromosome
(B) Complete fertility in the affected individuals
(C) Association with a lethal coat color mutation
(D) Being SRY-negative in the vast majority of cases

2013

87. A true hermaphrodite is strictly defined in developmental genetics as an individual possessing:
(A) Only completely ambiguous external genitalia
(B) Both ovarian and testicular tissues
(C) Testes alongside typical female external genitalia
(D) Ovaries alongside typical male external genitalia

2022

88. Which reproductive hormone, normally secreted by fetal testes, is responsible for the regression of female internal reproductive tracts (Müllerian ducts)?
(A) Estrogen steroid hormones
(B) Testosterone male hormone
(C) Anti-Müllerian Hormone
(D) Progesterone female hormone

2007

89. Consider the following statements regarding sex reversal syndromes:
I. SRY is the only gene involved in mammalian sex determination.
II. XX sex reversed dogs are typically fertile.
III. Polled Intersex Syndrome in goats is strongly linked to the polled phenotype.
Which of the above statements are incorrect?
(A) II and III
(B) I and III
(C) I
(D) I and II

2023

90. Which of the following genetic phenomena occurs when an offspring inherits both copies of

Downloaded from www.pdf.tube

2015

127. The pathogenesis of freemartinism involves the exchange of which crucial components through placental anastomoses?
(A) Maternal immunoglobulins and fetal erythrocytes
(B) Hematopoietic cells and Anti-Müllerian hormone
(C) Placental growth factors and nutritional fluids
(D) Free circulating XIST RNA and mitochondrial DNA

2002

128. What is the typical anatomical appearance of the reproductive tract of a freemartin heifer?
(A) Completely normal ovaries with bilateral absence of the vaginal canal
(B) Fully developed uterus and oviducts accompanied by a closed vulva
(C) Complete physical development of dual male and female functional organs
(D) Hypoplastic ovaries, underdeveloped uterus, and seminal vesicles

2005

129. How does hematopoietic chimerism manifest in a freemartin calf?
(A) The presence of both XX and XY hematopoietic cells
(B) The selective production of maternal erythrocytes
(C) A severe postnatal reduction in white blood cells
(D) The production of autoantibodies against blood cells

2005

130. While freemartinism is extremely common in cattle, it is relatively rare in sheep and pigs. Why?
(A) Sheep and pigs rarely carry twin pregnancies or large litters to full term.
(B) Endogenous fetal hormones do not control female reproductive tract development.
(C) Placental vascular fusions occur very rarely in sheep and pigs.
(D) The sex-determining region Y (SRY) gene functions differently in litters.

2022

131. Which diagnostic test is most definitively used to confirm freemartinism in a newborn female calf?
(A) PCR detection of Y-chromosome sequences in a blood sample
(B) Quantitative measurement of circulating blood estrogen levels
(C) Detailed visual inspection of specific coat color patterns
(D) A comprehensive diagnostic complete blood count profile

2023

132. What is the reproductive potential of the male co-twin of a freemartin calf?
(A) They are completely and irreversibly sterile at puberty.
(B) They invariably suffer from profound testicular hypoplasia.
(C) They possess a combination of ovarian and testicular tissues.
(D) Usually fertile, but may have reduced semen quality.

2016

133. The suppression of Müllerian duct development in the freemartin female is primarily attributed to the action of:
(A) High concentrations of estrogen from maternal circulation
(B) Anti-Müllerian Hormone (AMH) from the male co-twin
(C) Progesterone produced by the female's immature ovaries
(D) Luteinizing hormone (LH) from the male twin's pituitary

NODIA

2003

134. Consider the following statements concerning monogenic disorders in cattle:
I. BLAD is inherited as an autosomal dominant trait.
II. CVM causes severe vertebral malformations and abortions.
III. BLAD prevents leukocytes from migrating to infection sites.
IV. Both BLAD and CVM are primarily found in the Holstein breed.
V. CVM is caused by a mutation in the CD18 gene.
Which of the above statements are INCORRECT?
(A) II and III
(B) I and IV
(C) I and V
(D) II, IV and V

2009

135. Bovine Leukocyte Adhesion Deficiency (BLAD) is fundamentally caused by a mutation in the gene encoding which critical protein?

environment
(C) Genes located exclusively on the sex chromosomes
(D) A single gene that affects multiple independent traits

2003
154. Canine hip dysplasia is considered a multifactorial trait because its expression depends on:
(A) Only a single dominant mutation
(B) Interaction of multiple loci with diet and exercise
(C) Purely environmental factors with no genetic basis
(D) A chromosomal translocation combined with viral exposure

2024
155. Which of the following best describes a quantitative trait locus (QTL)?
(A) A DNA region associated with a continuously varying trait
(B) A unique nucleotide polymorphism that directly causes a disease
(C) The physical location of a centromere on a specific chromosome
(D) A regulatory sequence of DNA that strictly suppresses transcription

2016
156. In the context of canine hip dysplasia, what does the concept of "heritability" measure?
(A) The strict probability that an offspring will inherit the exact phenotype of the parent
(B) The number of genes involved in the disease
(C) The proportion of phenotypic variance in a population attributable to genetic variance
(D) The specific environmental trigger causing the dysplasia

2016
157. To reduce the incidence of a polygenic disorder like canine hip dysplasia in a breeding population, the most effective genetic strategy relies on:
(A) A simple single-gene DNA test to cull all carriers
(B) Breeding only animals that have been housed in small cages
(C) Inducing mutations to reverse the defective alleles
(D) Estimating breeding values based on phenotypic evaluations (e.g., radiograph scores) of the individual and its relatives

2022
158. The threshold model is often used to describe multifactorial diseases. According to this model, an animal manifests the clinical disease when:
(A) It inherits at least one dominant allele.
(B) It reaches a specific age, regardless of genetics.
(C) Its combined genetic liability and environmental exposure exceed a critical threshold point.
(D) All its chromosomes contain a mutation.

2019
159. Why is eradication of polygenic disorders significantly more difficult than monogenic recessive disorders?
(A) Polygenic disorders are universally lethal in utero.
(B) The exact combination of contributing alleles is usually unknown, and environmental factors mask the underlying genotype.
(C) The alleles causing polygenic disorders constantly undergo rapid mutation.
(D) Polygenic disorders do not impact animal welfare.

2001
160. Environmental modification (e.g., restricted caloric intake during growth) is often recommended for dogs genetically predisposed to hip dysplasia. This intervention demonstrates the principle of:
(A) Epistasis
(B) Incomplete penetrance
(C) Pleiotropy
(D) Gene-environment interaction

2015
161. Consider the following statements concerning lethal genes:
I. A true lethal gene causes death before the individual reaches reproductive age.
II. Sub-lethal genes cause death strictly during the embryonic stage.
III. Semi-lethal genes allow more than 50% but less than 100% of affected individuals to die before reproduction.
Which of the above statements are correct?
(A) I and III
(B) I and II
(C) I
(D) II and III

2025
162. How is a fully lethal genetic allele defined in population genetics?

Click Here to Buy book on Flipkart

2023
144. Deficiency of Uridine Monophosphate Synthase (DUMPS) is a genetic disorder affecting which crucial biochemical pathway?
(A) Purine nucleotide biosynthesis
(B) Urea cycle metabolism
(C) Anaerobic glycolysis pathway
(D) Pyrimidine biosynthesis pathway

2016
145. What is the phenotypic consequence for a dairy cow that is heterozygous (a carrier) for the DUMPS mutation?
(A) Appears normal but excretes high levels of orotic acid
(B) Develops severe megaloblastic anemia leading to early death
(C) Suffers from frequent, life-threatening bacterial infections
(D) Exhibits complete physical sterility and ovarian hypoplasia

2021
146. Citrullinemia is an autosomal recessive disorder most notably observed in Holstein-Friesian cattle. Which biochemical cycle is directly disrupted by this disease?
(A) Krebs cycle
(B) Calvin cycle
(C) Urea cycle
(D) Cori cycle

2001
147. Citrullinemia in cattle is caused by a nonsense mutation in the gene encoding which enzyme?
(A) Argininosuccinate synthetase (ASS)
(B) Arginase enzyme in the liver
(C) Ornithine transcarbamylase (OTC)
(D) Carbamoyl phosphate synthetase (CPS-I)

2012
148. The biochemical disruption in Citrullinemia leads to the toxic accumulation of which compound in the blood and brain of affected calves?
(A) Uric acid
(B) Lactic acid
(C) Orotic acid
(D) Ammonia

2016
149. Calves born with homozygous Citrullinemia typically present with which clinical signs within the first week of life?
(A) Severe skeletal deformities and joint laxity
(B) Progressive neurological deficits ending in death
(C) Profound and irreversible systemic immunodeficiency
(D) Hemorrhagic diarrhea and severe respiratory distress

2014
150. A major reason genetic disorders like DUMPS and Citrullinemia are targeted for eradication is because they cause economic losses primarily through:
(A) Decreased meat quality
(B) Increased veterinary costs for lifelong treatments
(C) Unexplained abortions and early calf mortality
(D) Zoonotic transmission to humans

2019
151. Why are molecular tests (like PCR-RFLP) preferred over biochemical assays (like measuring blood ammonia) for identifying Citrullinemia in a breeding herd?
(A) Biochemical assays are completely inaccurate.
(B) Molecular tests cure the mutation in the DNA.
(C) Blood ammonia is only elevated in carriers, not affected individuals.
(D) Molecular tests can accurately identify healthy, asymptomatic carriers.

2012
152. Match the monogenic disorder listed in List I with its affected gene or enzyme in List II:
List I: (Monogenic disorder)
P. BLAD
Q. Citrullinemia
R. DUMPS
S. CVM
List II: (Affected gene or enzyme)
1. SLC35A3
2. UMP Synthase
3. CD18 (beta-2 integrin)
4. Argininosuccinate synthetase
(A) P-3, Q-4, R-2, S-1
(B) P-4, Q-3, R-2, S-1
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-3, S-2

2002
153. Polygenic inheritance differs from monogenic inheritance in that polygenic traits are controlled by:
(A) A single gene with multiple alleles
(B) Multiple additive genes, often influenced by

Downloaded from www.pdf.tube

defects.

2013

118. A tetragametic chimera is produced through which specific embryological event?
(A) Symmetrical division of a single diploid embryo into monozygotic twins
(B) Polyspermic fertilization of a single viable ovum by two sperm cells
(C) Fusion of two independent dizygotic embryos early in gestation
(D) Abnormal retention of a polar body that is subsequently fertilized

2003

119. In cases of natural tetragametic chimerism involving a male and a female embryo, the resulting animal will typically exhibit:
(A) A completely normal and fully fertile male phenotype
(B) A completely normal and fully fertile female phenotype
(C) Severe generalized dwarfism and skeletal dysplasias
(D) Ambiguous external genitalia or true hermaphroditism

2002

120. Blood chimerism in domestic cattle is most frequently associated with which naturally occurring phenomenon?
(A) Twin pregnancies sharing placental blood vessels
(B) Severe inbreeding depression within closed herds
(C) Persistent viral infections during active gestation
(D) Reciprocal autosomal chromosomal translocations

2025

121. When fraternal twin calves (one male, one female) share a placental blood supply, the female calf is highly likely to become a:
(A) Tortoiseshell
(B) Chimera and a freemartin
(C) Tetraploid
(D) Carrier of an autosomal tissue trisomy

2014

122. Microchimerism refers to:
(A) The complete physical fusion of two microscopic pre-blastocyst embryos
(B) A small population of cells derived from another individual
(C) The exclusive localization of foreign DNA within the mitochondria
(D) A rare mutational state where only a single gene differs between tissues

NODIA

2023

123. The diagnostic confirmation of true tetragametic chimerism often requires:
(A) DNA profiling showing over two alleles at multiple loci in tissue samples
(B) A standard peripheral blood smear analyzed under high-power microscopy
(C) Sequential measurements of circulating serum testosterone concentrations
(D) Routine cytogenetic karyotyping displaying balanced chromosomal translocations

2021

124. Why might a chimera exhibit patches of different colored hair or skin?
(A) Random inactivation of X chromosomes leading to localized patchiness in female skins
(B) Temperature-dependent enzyme activity altering melanin synthesis in outer extremities
(C) The hair follicles derived from two genetically distinct zygotic cell lineages
(D) Highly localized spontaneous somatic mutations occurring in basal epidermal cells

2022

125. Select the correct sequence of embryological events leading to freemartinism in cattle:
(A) Dizygotic twinning → Placental vascular anastomosis → Transfer of male hormones/cells → Masculinization of the female tract
(B) Monozygotic twinning → Transfer of male hormones/cells → Placental vascular anastomosis → Masculinization of the female tract
(C) Placental vascular anastomosis → Dizygotic twinning → Masculinization of the female tract → Transfer of male hormones/cells
(D) Dizygotic twinning → Masculinization of the female tract → Transfer of male hormones/cells → Placental vascular anastomosis

2025

126. A freemartin is defined as a:
(A) Sterile male calf born co-twin to a female
(B) Fertile female calf born co-twin to a female
(C) Sterile female calf born co-twin to a male
(D) Calf exhibiting a chromosomal translocation

(A) An allele that prevents the animal from developing a specific coat color
(B) An allele that causes minor phenotypic abnormalities but allows normal reproduction
(C) An allele that causes the death of the organism, invariably reducing its evolutionary fitness to zero
(D) An allele that only causes death if inherited from the mother

2025
163. Some lethal alleles are described as dominant lethal. Why are dominant lethal mutations rarely transmitted across generations?
(A) Affected individuals die before they can reproduce.
(B) They are repaired instantly by cellular DNA proofreading.
(C) They are completely masked by wild-type recessive alleles.
(D) They only trigger lethality in the homozygous condition.

2012
164. A genetic defect causes the death of approximately 20% of affected calves within their first year, while the remaining 80% survive and can eventually reproduce. This gene would be classified as:
(A) Lethal
(B) Semi-lethal
(C) Beneficial
(D) Sub-lethal

2003
165. The presence of a recessive lethal allele in a population is usually maintained by:
(A) Complete directional selection against the dominant phenotype
(B) Normal heterozygous carriers who reproduce successfully
(C) High rates of spontaneous mutation at that specific locus
(D) Strict assortative mating patterns among affected animals

2001
166. A mutation that only causes death under certain environmental conditions (e.g., extreme temperature or specific diet) is known as a:
(A) Delayed lethal mutation
(B) Dominant lethal mutation
(C) Conditional lethal mutation
(D) Gametic lethal mutation

2023
167. A gametic lethal mutation primarily exerts its effect by:
(A) Causing the death of the newly formed zygote
(B) Preventing proper pairing of homologous chromosomes
(C) Arresting development at the early blastocyst stage
(D) Rendering gametes non-viable or unable to fertilize

2024
168. Some alleles are lethal in the homozygous state but produce a distinct, desirable phenotype in the heterozygous state (e.g., the crested phenotype in ducks). This specific genetic situation is an example of:
(A) Overdominance with recessive lethality
(B) Codominance with incomplete penetrance
(C) Epigenetic genomic paternal imprinting
(D) Variable expressivity and gene silencing

2011
169. What is the classic phenotypic presentation of a "Bulldog calf" in the Dexter cattle breed?
(A) Extremely tall and leggy stature accompanied by a narrow chest
(B) Severe achondroplasia, shortened limbs, and facial dysmorphia
(C) Normal skeletal cartilage development but a complete lack of hair
(D) An enlarged hypertrophied heart causing rapid failure at birth

2010
170. What is the expected phenotypic ratio of offspring at birth/conception from the mating of two heterozygous short-legged Dexter cattle (Short-legged : Normal long-legged : Bulldog calf)?
(A) 1:2:1
(B) 2:1:1
(C) 3:1:0
(D) 1:1:1

2011
171. The genetic condition causing the Bulldog calf anomaly in Dexter cattle is inherited as a:
(A) Recessive lethal trait with incomplete dominance
(B) Fully dominant autosomal maternal lethal trait
(C) Sex-linked recessive trait on the X chromosome
(D) Maternally inherited active mitochondrial

Click Here to Buy book on Amazon

31. (D) A normal female karyotype with 60 chromosomes
In bovine cytogenetics, the designation indicates the total diploid chromosome number and the sex chromosome complement of the animal. A count of sixty chromosomes represents the normal diploid number for this species, and the presence of two X chromosomes indicates a female, establishing that this is a normal female karyotype.

32. (C) C-banding
C-banding is a specialized cytogenetic staining technique used to visualize constitutive heterochromatin. This method involves treating chromosome spreads with an acid, followed by an alkali, and then staining with Giemsa. This selectively stains the highly repetitive DNA sequences located predominantly at the centromeres and on some sex chromosomes.

33. (B) p
In international cytogenetic nomenclature, the short arm of a chromosome is officially designated by the lowercase letter p, which originates from the French word petit, meaning small. The corresponding long arm of the chromosome is designated by the lowercase letter q, which simply follows p in the alphabet.

34. (D) From the centromere outward to the telomeres
In standard chromosome ideograms, numbering of the regions and individual bands is conducted systematically. The numbering always initiates at the centromere and proceeds outward along each arm toward the respective telomeres. This uniform system ensures that lower numbers represent proximal bands, while higher numbers designate distal bands.

35. (A) Cell culture → Colchicine treatment → Hypotonic treatment → Fixation → Staining
Preparing a metaphase spread begins with cell culture to induce active cell division. Colchicine is added to arrest dividing cells in metaphase. A hypotonic solution is then applied to swell the cells and spread the chromosomes. Finally, chemical fixation stabilizes the cellular structures before staining is performed to visualize the bands.

36. (C) Nondisjunction during meiosis or mitosis
Nondisjunction is the failure of homologous chromosomes or sister chromatids to separate properly during meiotic or mitotic nuclear division. This error results in daughter cells inheriting an abnormal number of chromosomes, either receiving an extra chromosome or lacking one entirely, which is the direct cause of numerical aneuploidy.

37. (A) Monosomy
Monosomy is a form of aneuploidy characterized by the loss of a single chromosome from a homologous pair in a diploid organism, represented mathematically as $2n-1$. This results in a total chromosome count that is one less than the normal diploid number, often leading to severe developmental imbalances.

38. (D) Two will be $n+1$ and two will be $n-1$
When a homologous chromosome pair fails to separate during the first meiotic division, all resulting gametes will be genetically abnormal. Two gametes will receive an extra chromosome, resulting in an $n+1$ complement. The other two gametes will lack that specific chromosome entirely, resulting in an $n-1$ complement after meiosis.

39. (B) Loss of both chromosomes of a homologous pair
Nullisomy is an extreme type of numerical chromosomal abnormality represented by the mathematical formula $2n-2$. This condition involves the complete loss of an entire homologous pair of chromosomes from the diploid set. In diploid organisms, nullisomy is almost universally lethal during early embryonic development due to the total absence of essential genes.

40. (C) It involves the gain of a homologous chromosome pair.
Tetrasomy is a specific type of numerical aneuploidy characterized by the presence of four copies of a particular chromosome rather than the normal homologous pair, represented as $2n+2$. This condition occurs due to the gain of an extra homologous chromosome pair, which is distinct from polyploidy where entire haploid sets are duplicated.

NODIA

(B) Arabian
(C) Clydesdale
(D) Quarter Horse

2009

182. Foals born with SCID typically appear normal at birth. Why do they inevitably succumb to overwhelming infections within the first few months of life?
(A) They possess a defective epidermis that allows bacterial entry.
(B) Their mothers do not produce colostrum.
(C) They fail to produce functional B and T lymphocytes.
(D) They lack functional neutrophils.

2018

183. The molecular defect causing SCID in Arabian horses is a deletion in the gene encoding:
(A) Uridine monophosphate synthase enzyme complex
(B) Endothelin receptor type B membrane protein
(C) Beta-2 integrin cell surface adhesion protein
(D) DNA-dependent protein kinase catalytic subunit

2012

184. What crucial cellular mechanism is impaired by the DNA-PKcs mutation in Arabian horses with SCID?
(A) V(D)J recombination of antigen receptor genes
(B) Oxidative phosphorylation within mitochondria
(C) Myelin sheath formation in motor neurons
(D) Calcium release from the sarcoplasmic reticulum

2008

185. Overo Lethal White Syndrome in horses presents a clinical parallel to which known congenital condition in humans?
(A) Duchenne Muscular Dystrophy
(B) Hirschsprung's Disease
(C) Tay-Sachs Disease
(D) Hemophilia A

2012

186. Because carriers of the SCID mutation in Arabian horses exhibit absolutely no clinical signs, the trait is inherited in what specific Mendelian manner?
(A) Autosomal dominant
(B) Autosomal recessive
(C) X-linked recessive
(D) Mitochondrial

2005

187. Spider Lamb Syndrome (Ovine Hereditary Chondrodysplasia) is an autosomal recessive lethal/semi-lethal disorder prevalent in Suffolk and Hampshire sheep. It is characterized by:
(A) Severe skeletal deformities, elongated limbs, and spinal curvature
(B) Complete absence of wool
(C) Overgrowth of muscle tissue yielding a "double-muscled" appearance
(D) Webbing between the hooves

2014

188. These questions consist of two statements each, printed as Assertion and Reason. Answer the questions selecting the appropriate option.
Assertion (A): Porcine Stress Syndrome (PSS) can result in sudden death of pigs during transport or minor veterinary procedures.
Reason (R): A mutation in the RYR1 gene causes uncontrolled calcium release in muscle cells when the animal is stressed or exposed to halothane anesthesia.
(A) Both (A) and (R) are true and (R) is the correct explanation of (A).
(B) Both (A) and (R) are true but (R) is not the correct explanation of (A).
(C) (A) is true but (R) is false.
(D) (A) is false but (R) is true.

2014

189. The gene mutation responsible for Porcine Stress Syndrome (PSS) affects the Ryanodine Receptor 1 (RYR1), which functions as a:
(A) Voltage-gated sodium channel in central nervous system neurons
(B) Inward-rectifying potassium channel in myocardial cardiac muscle
(C) Active cystic fibrosis chloride channel in specialized epithelial cells
(D) Calcium release channel in skeletal muscle sarcoplasmic reticulum

2010

190. Why was the RYR1 mutation historically tolerated or even inadvertently selected for by swine breeders before its lethal consequences were fully understood?
(A) It provides robust biological resistance against classical swine fever viruses.
(B) It significantly increased average litter size and neonatal survival rates.
(C) Carriers produced highly muscled, lean

Downloaded from www.pdf.tube

trait

2006

172. The mutation responsible for Dexter achondroplasia (Bulldog calf) has been identified in which specific gene?
(A) FGFR3
(B) MSTN (Myostatin)
(C) ACAN (Aggrecan)
(D) CD18

2006

173. What developmental process is primarily disrupted by the aggrecan mutation in Dexter cattle?
(A) Systemic hematopoiesis process
(B) Skeletal myogenesis pathway
(C) Early embryonic neurogenesis
(D) Endochondral ossification process

2024

174. If a breeder wishes to completely avoid producing Bulldog calves while maintaining a herd, what mating strategy must be strictly avoided?
(A) Mating a long-legged animal to another long-legged animal
(B) Mating a short-legged Dexter to a long-legged kerry animal
(C) Mating a short-legged Dexter to another short-legged Dexter
(D) Mating genetically distant outcrossed breeds of beef cattle

2013

175. While Dexter cattle display achondroplasia as a heterozygote advantage/phenotype, what happens to homozygous normal (wild-type) individuals in this breed?
(A) They suffer embryonic death and fail to survive in utero.
(B) They exhibit a normal, long-legged Kerry-type phenotype.
(C) They possess a distinct sub-lethal short-legged trait.
(D) They suffer from a severe, fatal genetic immunodeficiency.

2012

176. Which of the following conditions in humans shares genetic and phenotypic similarities (such as dwarfism in heterozygotes and lethality in homozygotes) with the Dexter cattle anomaly?
(A) Cystic Fibrosis
(B) Sickle Cell Anemia
(C) Huntington's Disease
(D) Achondroplasia

2011

177. The lethal nature of the homozygous Aggrecan mutation in Dexter cattle manifests during which stage of the animal's life?
(A) Fetal development, causing abortion around 7 months
(B) Late adulthood, causing progressive joint degeneration
(C) Puberty, preventing development of secondary sex traits
(D) The first week of neonatal life due to respiratory arrest

2003

178. Overo Lethal White Syndrome (OLWS) in horses is primarily associated with which coat color pattern?
(A) Uniform solid black coat
(B) Appaloosa leopard spotting
(C) Frame overo pinto pattern
(D) Classic roan hair pattern

NODIA

2018

179. Consider the following statements regarding Overo Lethal White Syndrome (OLWS):
I. It is caused by a mutation in the EDNRB gene.
II. Homozygous foals are born completely white.
III. Affected foals suffer from intestinal aganglionosis.
IV. It is a completely curable condition with early surgical intervention.
Which of the above statements are correct?
(A) I, II and III
(B) I, II and IV
(C) II, III and IV
(D) I and III

2004

180. The primary cause of death in foals afflicted with Overo Lethal White Syndrome is:
(A) Severe combined immunodeficiency leading to systemic septic infection
(B) Congenital cardiac anomalies leading to acute post-natal heart failure
(C) Aplastic bone marrow anemia and inability to produce red blood cells
(D) Intestinal impaction from absent functional nerve ganglia in bowel

2021

181. Severe Combined Immunodeficiency (SCID) is a lethal genetic disorder most notably found in which breed of horse?
(A) Thoroughbred

performed. If the bull is a carrier (heterozygous), how many distinct DNA fragment sizes (bands) would typically appear on the electrophoresis gel compared to a homozygous normal animal?
(A) Fewer bands, as the mutant allele is fully digested
(B) The identical band counts, but with twice the thickness
(C) Zero bands, as carrier status prevents enzyme activity
(D) Bands from both the normal and mutant allele digests

2019

200. Taq polymerase is essential for the PCR process because:
(A) It remains stable and active at high denaturation temperatures.
(B) It actively breaks hydrogen bonds between bases at room temperature.`
(C) It synthesizes short RNA primers required to initiate replication.
(D) It acts as a restriction endonuclease to cleave genomic templates.

2014

201. Which molecular technique relies on the hybridization of fluorescently labeled DNA to probes fixed on a solid surface, commonly used for genomic selection?
(A) Sanger Sequencing
(B) SNP Microarray
(C) Southern Blotting
(D) Western Blotting

2012

202. Why has PCR-based genotyping largely replaced traditional progeny testing for identifying carriers of recessive lethal mutations?
(A) PCR technology is less accurate but significantly cheaper to perform.
(B) PCR identifies carriers at birth, avoiding costly test matings.
(C) Progeny test practices actively cause lethal alleles to spread faster.
(D) PCR directly edits and corrects mutated DNA during the reaction process.

2010

203. Carrier detection refers to the identification of phenotypically normal individuals who possess one copy of a recessive mutant allele. What is the standard notation for the genotype of a carrier?
(A) Heterozygous
(B) Homozygous dominant
(C) Homozygous recessive
(D) Hemizygous

2024

204. In pedigree analysis, a square symbol typically represents a:
(A) Female
(B) Carrier of unknown sex
(C) Male
(D) Deceased individual

2018

205. The primary goal of an eradication strategy for a lethal genetic defect in a livestock population is to:
(A) Rapidly eliminate all beneficial dominant alleles from the gene pool
(B) Introduce novel artificial mutations to counteract genetic defects
(C) Clone somatic tissues of affected animals to study chronic diseases
(D) Systematically identify and manage carrier animals in breeding

2009

206. Consider the following statements regarding eradication strategies for genetic diseases:
I. Molecular testing allows for 100% accurate identification of carriers for known mutations.
II. Culling all carriers immediately is always the most economical choice for every population.
III. Pedigree analysis can estimate the probability of an animal being a carrier.
IV. AI (Artificial Insemination) sires must be rigorously tested because they have a massive impact on allele frequency.
V. Genomic selection ignores recessive mutations.
Which of the above statements are correct?
(A) I, III and V
(B) II, IV and V
(C) I, III and IV
(D) I, II and IV

2023

207. Why is completely culling every identified carrier of a recessive defect sometimes detrimental to a breed, particularly if the breed has a small population size?
(A) Carrier animals always possess superior physical traits and high adaptive value.
(B) It reduces genetic diversity, increasing inbreeding and other defects.
(C) The recessive allele will spontaneously mutate back into a dominant lethal state.

Click Here to Buy book on Flipkart

individuals carry one normal allele and one mutant allele on their autosomes. Because the mutation is dominant, the presence of this single defective allele results in the full clinical expression of the inherited disorder.

11. (B) Affected females inherit the allele from both parents.
Under X-linked recessive inheritance, females possess two X chromosomes and require two copies of the mutant allele to express the phenotype. Consequently, an affected female must inherit one mutant X chromosome from her mother and another from her affected father. Affected males, however, are hemizygous and express the trait with one mutant allele.

12. (A) 1 homozygous normal : 2 heterozygous carriers
Mating two heterozygous carriers yields a typical genetic ratio of one homozygous normal, two heterozygous carriers, and one homozygous affected individual. Because the homozygous affected state is prenatally or neonatally lethal, these individuals do not survive. The resulting genotypic ratio among the remaining survivors is one homozygous normal to two heterozygous carriers.

13. (A) Autosomal recessive
Autosomal recessive disorders require two copies of the mutated allele for clinical expression. Heterozygous individuals possess one mutant and one normal allele, remaining clinically unaffected carriers. The trait can remain hidden for multiple generations, appearing only when two carrier individuals mate and both transmit their defective allele to a single offspring.

14. (C) X-linked dominant
In X-linked dominant inheritance, affected males transmit their single mutated X chromosome to all of their daughters, who will inevitably express the disease. Since fathers pass only their Y chromosome to their sons, none of their male offspring will inherit the paternal X-linked mutation or exhibit the associated clinical symptoms.

15. (D) The first affected individual evaluated in the family.
In medical and veterinary genetics, the proband designates the individual who first brings a particular family pedigree to the attention of investigators or clinicians. This subject serves as the starting point for tracing the inheritance pattern of a genetic trait or disease within a lineage, regardless of whether they are alive.

16. (C) It increases the probability of homozygosity by descent.
Inbreeding involves the mating of closely related individuals, which increases the likelihood that offspring will inherit identical alleles from a common ancestor. This process significantly raises the probability of homozygosity by descent. Consequently, rare recessive deleterious alleles are more frequently paired together, leading to a higher incidence of affected individuals.

17. (A) Both A and R are true and R is the correct explanation of A.
Mammalian males have one X and one Y chromosome, making them hemizygous for X-linked genes. A single recessive mutation on their sole X chromosome inevitably causes phenotypic expression of the disorder. Conversely, females have two X chromosomes and require two copies of the mutation, explaining why males are much more frequently affected.

18. (D) Genomic imprinting based on parental origin.
Classic Mendelian genetics assumes that alleles from both parents are expressed equally in the offspring. Genomic imprinting is an epigenetic exception where expression of specific genes depends entirely on which parent transmitted the allele. One allele is transcriptionally silenced via methylation during gametogenesis, leaving only the other parental allele active.

NODIA

19. (A) Penetrance
Penetrance is defined as the proportion of individuals in a population carrying a particular genetic variant who express the associated clinical phenotype. When all individuals with the genotype show the trait, penetrance is complete. If some carriers remain phenotypically normal, the trait exhibits incomplete penetrance, which is often influenced by genetic modifiers.

20. (C) Individuals with the same genotype show varying clinical severity.
Variable expressivity occurs when individuals carrying the exact same disease-causing genotype exhibit a range of different clinical signs and levels of severity. While the disease-

ANSWERS

1. (B) Nonsense mutation
Nonsense mutations occur when a single nucleotide substitution alters a codon that normally encodes an amino acid into a translation termination signal. This premature stop codon halts polypeptide synthesis prematurely, resulting in a truncated, typically nonfunctional protein product. This specific molecular event severely impacts gene expression without altering the reading frame of the transcribed mRNA molecule.

2. (D) DNA to RNA to Protein
The central dogma of molecular biology outlines the classical pathway of genetic information transfer within biological systems. Deoxyribonucleic acid serves as the primary template, which is first transcribed into messenger ribonucleic acid. This ribonucleic acid intermediate is subsequently translated into functional polypeptides, representing the unidirectional, standard flow of genetic material across living organisms.

3. (A) A shift in the reading frame of the ribosome
The addition or loss of a single nucleotide within a protein-coding sequence disrupts the triplet codon grouping. This alteration shifts the triplet reading frame downstream from the mutation site, completely changing the amino acid sequence during translation. Such structural modifications often result in premature stop codons, leading to the production of nonfunctional proteins.

4. (C) It always alters the total polypeptide length.
Point mutations refer to single nucleotide base changes, which can lead to silent, missense, or nonsense alterations. Silent point mutations do not alter the translated amino acid sequence, and missense mutations only change a single amino acid. Therefore, these specific genetic alterations do not always impact the overall length of the resulting synthesized polypeptide chain.

5. (A) Missense mutation
A missense mutation is a point mutation in which a single nucleotide change results in a codon that codes for a different amino acid. This amino acid substitution can alter the secondary or tertiary structure of the protein, potentially impacting its overall function, stability, and cellular activity without introducing a premature stop signal.

6. (D) Genetic code degeneracy
Genetic code degeneracy refers to the property where most of the twenty standard amino acids are specified by more than one distinct codon. This redundancy provides protection against point mutations, as a base change at the third position of a codon often results in a synonymous codon that translates into the exact same amino acid.

7. (B) A purine is replaced by a pyrimidine base.
A transversion represents a specific category of point mutation where a two-ring purine base is replaced by a single-ring pyrimidine base, or vice versa. This structural type of substitution contrasts with transition mutations, which only involve exchanges within the same chemical class of bases, such as purine-to-purine or pyrimidine-to-pyrimidine substitutions.

8. (C) P-4, Q-1, R-2, S-3
A point transition involves exchanging a purine for another purine, while a frameshift mutation shifts the reading frame. A nonsense mutation introduces a premature stop codon, halts translation early, and shortens the polypeptide. Conversely, a silent mutation changes the nucleotide sequence but results in no change to the corresponding amino acid due to code degeneracy.

9. (D) Frameshift mutations
Ethidium bromide is a planar intercalating agent that slides between adjacent nitrogenous base pairs of the deoxyribonucleic acid double helix. During replication, this distortion causes the DNA polymerase to insert or delete one or more nucleotides. This insertion or deletion results in a frameshift mutation, altering the translation reading frame of the gene.

10. (A) They will express the disease phenotype.
In autosomal dominant inheritance patterns, a single copy of the mutant allele is sufficient to manifest the disease phenotype. Heterozygous

NODIA

carcasses of high economic value.
(D) It allowed finishing pigs to mature and reach market weight twice as fast.

2015
191. The mutation causing Spider Lamb Syndrome has been mapped to the FGFR3 gene. This gene is critical for regulating:
(A) The synthesis of wool keratin structural proteins
(B) Bone growth via inhibition of chondrocytes
(C) Immune responses against bacterial lung pneumonia
(D) The enzymatic breakdown of glycogen in the liver

2022
192. Malignant Hyperthermia is an alternative clinical manifestation of the exact same genetic mutation that causes which of the following animal disorders?
(A) Bovine Leukocyte Adhesion Deficiency (BLAD)
(B) Severe Combined Immunodeficiency (SCID)
(C) Spider Lamb Syndrome
(D) Porcine Stress Syndrome (PSS)

2013
193. What is the typical condition of the meat post-mortem from a pig that has suffered a PSS crisis prior to slaughter?
(A) It becomes pale, soft, and exudative (PSE).
(B) It becomes excessively dark, firm, and dry (DFD).
(C) It turns green due to rapid bacterial overgrowth.
(D) It is entirely unaffected.

2004
194. The definitive non-invasive molecular diagnosis to eliminate Spider Lamb Syndrome from a breeding flock relies on identifying:
(A) The physical manifestation of skeletal deformities
(B) High concentrations of ionized calcium in blood
(C) The T to A FGFR3 gene mutation using PCR
(D) The presence of dominant alleles via test crosses

2015
195. Polymerase Chain Reaction (PCR) is a foundational tool in molecular diagnostics. What is its primary function?
(A) To exponentially amplify target DNA
(B) To sequence complete genomic templates
(C) To translate coding DNA into proteins
(D) To cleave DNA at palindromic sequences

2013
196. Restriction Fragment Length Polymorphism (RFLP) is often coupled with PCR to detect mutations. How does RFLP distinguish between a mutant and a normal allele?
(A) By selectively amplifying only the specific mutant allele sequences
(B) By measuring the molecular weight of intact genomic DNA templates
(C) By attaching targeted fluorescent probes exclusively to mutant DNA
(D) By using restriction enzymes that cut only one of the alleles

2022
197. Match the molecular diagnostic tool listed in List I with its specific application or mechanism in List II:
List I: (Molecular diagnostic tool)
P. PCR
Q. RFLP
R. SNP Array
S. Karyotyping
List II: (Application or mechanism)
1. Detects large chromosomal rearrangements
2. Amplifies specific DNA segments
3. Genotypes thousands of genetic markers simultaneously
4. Differentiates alleles based on restriction enzyme cutting
(A) P-1, Q-4, R-3, S-2
(B) P-2, Q-4, R-3, S-1
(C) P-2, Q-3, R-4, S-1
(D) P-3, Q-2, R-1, S-4

2007
198. Single Nucleotide Polymorphism (SNP) array genotyping is highly advantageous for modern animal breeding because it allows for:
(A) Direct gene editing and therapeutic correction of mutations in live animals
(B) Visualizing physical chromosomal structures using high-power light microscopy
(C) Simultaneous genotyping of thousands of markers across the genome
(D) Artificially inducing highly favorable genetic mutations using radiation

2014
199. A breeder wants to test their bull for the presence of the BLAD mutation. A PCR-RFLP test is

Downloaded from www.pdf.tube

causing mutation penetrance may be complete, the actual physiological manifestations can vary from extremely mild to life-threatening due to environmental factors and genetic background.

21. (A) Pleiotropy
Pleiotropy is a genetic phenomenon where a single gene mutation controls or influences multiple, seemingly unrelated anatomical or physiological traits. In connective tissue disorders like Marfan syndrome, a mutation in a single gene encoding a structural protein leads to diverse clinical abnormalities across the skeletal, ocular, and cardiovascular systems simultaneously.

22. (D) Independent assortment
Penetrance refers to the likelihood that a specific genotype will produce its associated phenotype. This is influenced by epigenetic modifications, environmental conditions, and modifier genes that alter gene expression. Independent assortment is a meiotic process governing allele transmission to gametes, which has no effect on whether an inherited genotype is phenotypically expressed.

23. (B) Incomplete penetrance
Incomplete penetrance describes a genetic situation where less than one hundred percent of individuals carrying a specific disease-causing or trait-associated genotype display the expected clinical phenotype. In this scenario, only eighty percent of the livestock with the dominant gene develop horns, while twenty percent remain hornless despite carrying the gene.

24. (C) The Merle gene causing coat patterning and deafness in dogs.
Pleiotropy occurs when a single genetic locus controls multiple distinct, seemingly unrelated phenotypic traits in an animal. The Merle gene in dogs is a classic example because this single mutation simultaneously alters coat pigmentation patterns and disrupts the development of the inner ear, leading to pigment dilutions and congenital deafness.

25. (D) Clinical signs of the disorder manifest later in life.
Age-dependent penetrance is a genetic phenomenon in which the clinical signs and symptoms of an inherited disorder do not appear at birth. Instead, the likelihood of expressing the disease phenotype increases as the individual grows older, with clinical manifestation occurring later in life after a prolonged asymptomatic period.

26. (A) II and III
Pleiotropy is correctly defined as a single gene locus affecting multiple distinct phenotypic traits. Incomplete penetrance occurs when some individuals carrying a specific genotype fail to express any corresponding clinical phenotype. In contrast, penetrance refers to the proportion of individuals expressing a phenotype, whereas the varying severity of that phenotype is called expressivity.

27. (B) Colchicine
Colchicine is an alkaloid compound widely used in cytogenetics to prepare karyotypes. It works by binding to tubulin dimers, which effectively prevents the assembly of microtubules and disrupts mitotic spindle fiber formation. Consequently, dividing cells are arrested during metaphase, when chromosomes are at their most condensed and visible state.

28. (B) G-banding
G-banding, or Giemsa banding, is the standard cytogenetic staining technique used to visualize chromosomes. Chromosomes are treated with trypsin to digest associated proteins and then stained with Giemsa dye. This process produces a highly characteristic pattern of alternating dark and light bands, enabling the identification of individual homologous chromosomes.

NODIA

29. (A) AT-rich, late replicating heterochromatin
In G-banded chromosomes, the dark bands correspond to regions that are highly condensed, rich in adenine and thymine nucleotides, and late-replicating during the cell cycle. These regions contain heterochromatin, which is transcriptionally inactive. Conversely, light bands represent GC-rich, gene-dense euchromatin that replicates early and is transcriptionally active.

30. (C) Karyotype
A karyotype is the prepared photographic representation of the complete set of chromosomes within a somatic cell. It involves organizing the chromosomes into matching homologous pairs and arranging them systematically in descending order of physical size, centromere position, and banding pattern, allowing clinicians to detect chromosomal abnormalities.

(D) It triggers a stress-induced immediate increase in the germline mutation rate.

2019

208. An alternative strategy to wholesale culling of carriers in a valuable genetic line is:

(A) Mating heterozygous carriers only to other confirmed heterozygous carriers

(B) Highly intensive inbreeding of carrier animals to isolate genetic defects

(C) Utilizing all carrier lines for commercial meat production and processing

(D) Mating carriers to clear animals and selecting only clear offspring

2013

209. A sire is suspected of being a carrier for a rare autosomal recessive lethal defect for which no DNA test exists. What is the traditional method to prove with high statistical confidence (e.g., 99%) that he is clear?

(A) Mate him to his own daughters or carrier females and observe progeny

(B) Mate him to a completely random sample of unrelated healthy dairy cows

(C) Perform a high-resolution cytogenetic karyotype analysis on his blood

(D) Feed him a highly specialized metabolic diet and monitor daily health

2014

210. The rapid global spread of genetic defects like BLAD and CVM in dairy cattle underscores the critical importance of:

(A) Severely restricting the international trade of all animal germplasm products

(B) Transitioning dairy herds entirely back to natural service mating systems

(C) Mandatory genetic screening of elite AI sires before semen distribution

(D) Utilizing only closed, highly inbred purebred lines for milk production

****

Click Here to Buy book on Amazon

41. (D) Unmasked lethal recessive alleles and haploinsufficiency.
Autosomal monosomy is typically lethal because having only a single copy of an autosome exposes any deleterious recessive mutations present on that remaining chromosome. Furthermore, the loss of half the normal gene dosage for multiple critical developmental genes, a phenomenon known as haploinsufficiency, prevents normal embryonic development, leading to early mortality.

42. (A) Severe developmental imbalances lead to early embryonic mortality in animals.
Autosomal trisomy introduces a third copy of a chromosome, disrupting the delicate genetic balance required for normal development. In domestic animal species, these genetic imbalances are highly detrimental, triggering early embryonic or fetal mortality in utero. Consequently, affected individuals rarely survive to birth, making live-born cases exceptionally rare compared to humans.

43. (B) Nondisjunction at Meiosis I
During normal male spermatogenesis, the X and Y chromosomes pair and then segregate into separate cells during the first meiotic division. If nondisjunction occurs at this stage, both sex chromosomes migrate together into the same daughter cell. Subsequent normal second meiotic division yields gametes containing both an X and a Y chromosome.

44. (B) I and III
Nondisjunction occurring during the second meiotic division results in two normal gametes, one gamete with an extra chromosome, and one gamete lacking that chromosome. Monosomy describes a state of having one missing chromosome, denoted as $2n-1$. However, trisomy is an aneuploid state rather than a form of balanced euploidy.

45. (A) It has three or more complete chromosome sets.
Polyploidy is a chromosomal condition in which an organism possesses three or more complete, homologous sets of chromosomes in its somatic cells. This condition differs from aneuploidy, which involves the loss or gain of individual chromosomes, and from diploidy, which is characterized by having exactly two complete sets of chromosomes.

NODIA

46. (C) Dispermy, where two sperm fertilize one ovum.
In mammals, triploidy occurs when a somatic cell contains three haploid sets of chromosomes. The most common cause is dispermy, a fertilization event where two distinct, normal haploid sperm cells simultaneously penetrate and fertilize a single normal haploid egg cell. This results in a zygote with three sets of chromosomes.

47. (D) It is usually lethal during early embryonic stages.
In domesticated mammalian species, complete duplication of chromosome sets disrupts critical genomic imprinting and dose-dependent embryonic gene expression networks. This catastrophic imbalance prevents normal organogenesis and placental development. Consequently, triploidy and tetraploidy are highly deleterious conditions that almost always result in early embryonic mortality in utero.

48. (C) Involve whole haploid sets of chromosomes.
Euploidy defines a chromosomal state where the total chromosome number is an exact integral multiple of the basic haploid set characteristic of the species. This type of variation involves the loss or gain of entire sets of chromosomes, as seen in monoploidy, diploidy, triploidy, and other polyploid states.

49. (B) Polyploidy
Endoreduplication is a specialized cellular event in which chromosomes replicate their DNA content during the synthesis phase, but the cell subsequently fails to undergo mitosis or cytokinesis. This replication without division doubles the chromosome number within the cell, directly leading to the formation of polyploid cell lines or organisms.

50. (D) Sterility, diverting energy to somatic growth.
In aquaculture, triploid fish and shellfish possess three sets of chromosomes, which prevents homologous pairing during meiosis and leads to functional sterility. Because sterile animals do not expend energy on gonad development or reproductive behavior, they divert their metabolic resources entirely to somatic growth, yielding larger and high-quality market products.

51. (A) Triploidy
During normal oogenesis, meiosis produces a haploid egg and throws off polar bodies containing

72. (C) XO
Turner's syndrome is a sex chromosome monosomy characterized by a single X chromosome and no second sex chromosome, designated as XO. This condition affects female animals, who have a diploid chromosome count of $2n-1$. Affected individuals typically exhibit gonadal dysgenesis, severely underdeveloped reproductive tracts, and complete infertility.

73. (D) Gonadal dysgenesis
In domestic animals like mares, the XO genotype leads to gonadal dysgenesis, characterized by small, inactive, and fibrous streak ovaries. Because of the lack of proper ovarian development and estrogen secretion, these mares show irregular or absent estrus cycles and are sterile, although their external genitalia appear phenotypically female.

74. (A) XXY
Klinefelter's syndrome is a sex chromosome aneuploidy in male mammals characterized by the presence of an extra X chromosome, resulting in an XXY karyotype. Affected individuals are phenotypically male due to the presence of the Y chromosome, but they suffer from testicular hypoplasia, azoospermia, and complete sterility throughout their adult lives.

75. (B) XXY
The gene for coat color in cats is carried on the X chromosome, with different alleles producing orange or black pigment. Expressing both colors requires two X chromosomes. Male cats are typically XY and cannot be tortoiseshell, but those with Klinefelter's syndrome (XXY) can express both colors while remaining sterile males.

76. (B) Testicular hypoplasia and lack of sperm.
The presence of an extra X chromosome in XXY males disrupts germ cell development in the seminiferous tubules. This disruption causes severe testicular hypoplasia and prevents spermatogenesis, leading to azoospermia. While the Y chromosome directs male phenotypic development, the testicular tissue remains underdeveloped, resulting in absolute infertility.

77. (A) Turner's-like syndrome (XO)
Turner's-like syndrome, characterized by an XO karyotype, is the primary sex chromosome monosomy in female domestic animals. Affected females lack a second sex chromosome, which is necessary for normal ovarian development. Consequently, they display small stature, complete failure to undergo estrus, and hypoplastic, non-functional streak ovaries throughout life.

78. (C) Phenotypically male anatomy.
The Y chromosome carries the SRY gene, which is the master switch for male sex determination in mammals. Even in the presence of two X chromosomes, the activation of the SRY gene drives the undifferentiated embryonic gonads to develop into testes. This ensures the individual develops a phenotypically male anatomy.

79. (D) Paternal Meiosis II
The XYY karyotype requires a sperm cell carrying two Y chromosomes. This condition is caused by a nondisjunction event of the sister chromatids of the Y chromosome during the second meiotic division of paternal spermatogenesis. Fertilization of a normal haploid egg by this YY sperm results in an XYY offspring.

80. (C) 1, 3, 4, 2
The pathogenesis of XXY syndrome begins with a meiotic nondisjunction event in a parent's gamete. This abnormal gamete participates in fertilization, creating an XXY zygote. During development, the SRY gene on the Y chromosome initiates testis differentiation, but the extra X chromosome ultimately causes testicular hypoplasia during sexual maturation.

81. (B) On the short arm of the Y chromosome
The sex-determining region Y gene, or SRY, is the primary genetic trigger for male sexual development in mammals. Under normal conditions, this gene is located on the short arm of the Y chromosome, outside the pseudoautosomal region. Its localized expression in the indifferent embryonic gonad initiates the pathway toward testis development.

82. (D) It initiates indifferent gonad development into a testis.
The SRY gene encodes a key transcription factor known as testis-determining factor. The primary role of this protein is to activate male-specific developmental pathways, such as the upregulation of SOX9. This cascade instructs the indifferent embryonic gonads to differentiate into testes rather than ovaries during early fetal development.

Downloaded from www.pdf.tube

site where the liberated segment is subsequently integrated.

62. (C) P-3, Q-1, R-2, S-4
Paracentric inversions occur entirely on one side of the centromere, leaving it outside the inverted segment. In contrast, pericentric inversions span the centromere. Deletions result in a loss of genetic material, whereas duplications lead to a repeated segment. These structural characteristics define and distinguish these chromosome aberrations from one another.

63. (D) Exchange of segments between two non-homologous chromosomes
A reciprocal translocation involves double-strand breaks occurring on two non-homologous chromosomes. The broken segments are then swapped and reattached to the opposite chromosomes. Because there is a mutual exchange of genetic material without any net loss or gain, the individual carrier typically remains phenotypically normal and balanced in their genome.

64. (C) Robertsonian translocation
The 1/29 translocation in cattle is a classic Robertsonian translocation. It involves the physical fusion of two non-homologous acrocentric chromosomes, specifically chromosomes one and twenty-nine, at their centromeres. This fusion produces a single, large metacentric chromosome, reducing the total chromosome count while keeping the genetic material balanced.

65. (B) Reduced fertility from embryonic mortality
Heterozygous carriers of the 1/29 Robertsonian translocation are phenotypically normal but exhibit reduced fertility. During meiosis, the rearranged chromosome forms a trivalent structure. This leads to the production of unbalanced, aneuploid gametes. Fertilization of these abnormal gametes results in nonviable embryos, causing early embryonic death and reduced reproductive rates.

66. (D) Two acrocentric or telocentric chromosomes
Robertsonian translocations are structural rearrangements restricted to acrocentric or telocentric chromosomes. The long arms of two non-homologous acrocentric chromosomes fuse at their centromeres to form a single metacentric chromosome. The short arms, containing repetitive ribosomal DNA, are typically lost without causing any clinical symptoms in the carrier.

67. (A) It reduces the total chromosome number by one.
In a balanced carrier of a Robertsonian translocation, two separate acrocentric chromosomes fuse to form a single, large metacentric chromosome. This fusion reduces the total chromosome count by one. However, because nearly all essential genetic information is retained on the fused long arms, the carrier remains phenotypically normal and healthy.

NODIA

68. (C) They form a cruciform (cross-shaped) quadrivalent.
Heterozygosity for a reciprocal translocation means two normal chromosomes and two translocated chromosomes must pair during prophase I of meiosis. To maximize homologous pairing, these four chromosomes align to form a characteristic cross-shaped structure known as a cruciform quadrivalent, which subsequently segregates in various patterns during anaphase I.

69. (A) Alternate segregation
During meiotic segregation in a reciprocal translocation heterozygote, alternate segregation occurs when diagonal chromosomes in the quadrivalent migrate to the same pole. This sends either both normal chromosomes or both translocated chromosomes into the gamete. These gametes carry a complete, balanced set of genetic material, resulting in viable offspring.

70. (D) Swedish Red and White
The 1/29 Robertsonian translocation was first discovered and characterized in the Swedish Red and White cattle breed. It has since been identified in numerous other beef and dairy cattle breeds worldwide, consistently causing a reduction in fertility due to early embryonic mortality in heterozygous cows mated to carrier bulls.

71. (B) I, II and IV
The 1/29 translocation involves the fusion of acrocentric chromosomes one and twenty-nine, which reduces the total chromosome number to fifty-nine in heterozygous carriers. Although carriers are phenotypically normal, they exhibit reduced fertility due to meiotic imbalances. Homozygotes are viable, and it is a Robertsonian, rather than a reciprocal translocation.

participates in the placental blood exchange, the introduction of female cells and hormones can occasionally alter testicular development, leading to a potential reduction in overall semen quality and sperm concentration at sexual maturity.

133. (B) Anti-Müllerian Hormone (AMH) from the male co-twin
The regression and regression of the female reproductive tract in a freemartin is initiated by anti-Müllerian hormone. Produced early in gestation by the testes of the male co-twin, this hormone crosses the placental vascular bridge into the female's bloodstream, actively binding to receptors in her target tissues to block the development of her Müllerian ducts.

134. (C) I and V
Bovine leukocyte adhesion deficiency is inherited as an autosomal recessive trait, not dominant. Additionally, complex vertebral malformation is caused by a missense mutation in the SLC35A3 gene, not the CD18 gene. Conversely, complex vertebral malformation causes severe spinal defects, bovine leukocyte adhesion deficiency impairs leukocyte migration, and both are prevalent in Holsteins.

135. (D) Beta-2 integrin (CD18)
Bovine leukocyte adhesion deficiency is an inherited autosomal recessive disorder caused by a single point mutation in the gene that encodes beta-2 integrin, also known as CD18. This membrane glycoprotein is a crucial component of adhesion molecules required for white blood cells to bind to vascular endothelium and migrate into infected tissues.

136. (A) Recurrent, severe bacterial infections and early death
Calves homozygous for the bovine leukocyte adhesion deficiency mutation exhibit a profoundly compromised immune system. Because their neutrophils cannot migrate to sites of infection, these animals suffer from recurrent, severe bacterial infections, persistent granulocytosis, periodontitis, and poor wound healing, which invariably lead to early death during the first year of life.

137. (B) A CD18 defect preventing neutrophil adhesion to blood vessel walls
In calves affected by bovine leukocyte adhesion deficiency, a missense mutation in the CD18 gene leads to a defective beta-2 integrin subunit. This structural defect prevents the functional assembly of leukocyte adhesion molecules. Consequently, circulating neutrophils are unable to adhere to the blood vessel walls, blocking their extravasation into tissues to fight pathogens.

138. (B) SLC35A3
Complex vertebral malformation is a lethal autosomal recessive disorder in Holstein cattle caused by a specific missense mutation in the SLC35A3 gene. This gene encodes a solute carrier family protein that is vital for normal cellular biochemical functions, and its impairment directly disrupts the structural development of the vertebral column during embryonic skeletogenesis.

139. (A) UDP-N-acetylglucosamine transport into the Golgi
The missense mutation in the SLC35A3 gene disrupts the function of the Golgi transporter protein responsible for import of UDP-N-acetylglucosamine. This transport defect impairs the normal glycosylation of proteins and lipids within the Golgi apparatus, which is essential for the synthesis of extracellular matrix components required for proper embryonic skeletal and cartilage development.

140. (C) Shortened neck, scoliosis, and bilateral arthrogryposis
Affected calves born with complex vertebral malformation present with a distinct cluster of skeletal anomalies. These include a shortened neck, severe scoliosis or hemivertebrae of the thoracic and lumbar spine, and bilateral arthrogryposis of the forelimbs. Most affected pregnancies end in premature abortion, while live-born calves are highly non-viable.

141. (D) Extensive use of heavily favored AI carrier sires
The high prevalence of both bovine leukocyte adhesion deficiency and complex vertebral malformation within Holstein herds is due to popular sire syndrome. Highly productive elite bulls, who happened to be asymptomatic heterozygous carriers, were intensively used worldwide through artificial insemination. This practice inadvertently disseminated the recessive mutant alleles throughout the global dairy gene pool.

Click Here to Buy book on Flipkart

83. (A) Polled trait (hornless)
Polled Intersex Syndrome is a genetic disorder in goats that is pleiotropically linked to the polled phenotype. The dominant allele causing hornlessness is associated with a genomic deletion. In homozygous females, this deletion causes disruption of ovarian development, leading to varying degrees of intersexuality and complete reproductive tract abnormalities.

84. (C) XX karyotype with intersex phenotype
In goats, Polled Intersex Syndrome selectively affects individuals that are genetically female, having an XX sex chromosome constitution, and are homozygous for the dominant polled allele. Although these animals are chromosomal females, the genetic deletion prevents normal ovarian development, causing them to develop an intersex phenotype with masculinized gonads.

85. (A) FOXL2 and PISRT1
The genetic cause of Polled Intersex Syndrome is a deletion of a regulatory region that controls the expression of FOXL2 and PISRT1. These genes are crucial for promoting ovarian pathway development and repressing male pathways in female embryos. Their downregulation leads to sex reversal in genetically female homozygous polled goats.

86. (D) Being SRY-negative in the vast majority of cases
XX sex reversal in canine breeds is a disorder of sexual development where chromosomal females develop testicular tissue. Unlike most mammalian sex reversals, which involve the translocation of the SRY gene, affected dogs are almost always SRY-negative. This indicates that other autosomal or X-linked genes in the sex-determining pathway are mutated.

87. (B) Both ovarian and testicular tissues
In developmental genetics, a true hermaphrodite is characterized by the coexistence of both ovarian and testicular tissues in the same individual. This can manifest as separate ovaries and testes, or as combined gonads known as ovotestes. This condition differs from pseudohermaphroditism, where the gonads match the chromosomal sex but external genitalia are ambiguous.

88. (C) Anti-Müllerian Hormone
Anti-Müllerian Hormone is a glycoprotein secreted by the Sertoli cells of the developing fetal testes. Its primary function during male sexual differentiation is to cause the active regression of the Müllerian ducts, which would otherwise develop into the uterus, fallopian tubes, and upper vagina, ensuring proper male internal reproductive anatomy.

89. (D) I and II
The SRY gene is the master regulator, but several other downstream genes are essential for mammalian sex determination. In addition, XX sex reversed dogs are infertile due to malformed reproductive tracts and abnormal gonadal tissue. Polled Intersex Syndrome in goats, however, is indeed strongly associated with the dominant hornless phenotype.

90. (B) Uniparental disomy
Uniparental disomy describes a situation where an individual inherits two copies of a chromosome from one parent and no copy from the other parent. This can occur when a trisomic zygote sheds one of the extra chromosomes early in development, a survival mechanism that occasionally leaves both remaining homologous chromosomes from a single parent.

91. (B) Maternal meiotic nondisjunction
Maternal meiotic nondisjunction is the most common cause of autosomal trisomies in abortuses. During oogenesis, homologous chromosomes fail to separate during Meiosis I, or sister chromatids fail to separate in Meiosis II. This error creates an egg with an extra chromosome, which, upon fertilization, results in a trisomic bovine zygote.

92. (B) Severe craniofacial, cardiac, and skeletal defects
Trisomy eighteen is a highly deleterious chromosomal abnormality in mammals that causes severe multi-system developmental defects. Affected individuals exhibit complex congenital anomalies, including characteristic craniofacial dysmorphism, structural cardiac malformations, and severe skeletal abnormalities. Most affected cases result in spontaneous abortion or early neonatal death due to these organ system failures.

93. (A) $2n + 1$
Autosomal trisomies, such as Trisomy sixteen, are characterized by the presence of a single extra chromosome in somatic cells. Instead of the

Downloaded from www.pdf.tube

typical homologous pair, there are three copies of chromosome sixteen. This numerical aneuploidy alters the diploid complement and is represented by the genetic formula $2n + 1$.

94. (C) Multiple congenital malformations
Autosomal trisomies cause significant gene dosage imbalances that disrupt embryonic development across multiple tissues. Although most cases are embryonic lethal, the rare animals that survive to birth consistently present with multiple congenital malformations, including craniofacial abnormalities, cardiac defects, and severe motor retardation, alongside drastically reduced lifespans in domestic species.

95. (D) It increases nondisjunction due to prolonged oocyte arrest in dictyotene.
Oocytes are arrested in the dictyotene stage of prophase I during fetal development and remain dormant until ovulation. As maternal age advances, the prolonged duration of this arrest degrades the cohesive proteins holding homologous chromosomes together. This structural degradation increases the frequency of meiotic nondisjunction during ovulation, resulting in trisomies.

96. (C) Gene dosage effects from triplicated chromosome overexpression.
The presence of an extra autosome results in three copies of all the genes located on that chromosome. This leads to the transcription and translation of fifty percent more product for these genes, disrupting delicate regulatory networks. These gene dosage imbalances interfere with normal cellular processes, causing severe multi-system developmental defects.

97. (B) Trisomy rescue
Trisomy rescue is a cellular event that occurs when an aneuploid zygote containing three copies of a chromosome undergoes chromosome loss during early mitotic divisions. This loss restores a diploid chromosome number in the daughter cells. If both remaining chromosomes happen to originate from the same parent, uniparental disomy occurs.

98. (A) Both A and R are true and R is the correct explanation of A.
Chromosomal mosaicism is characterized by the presence of multiple genetically distinct cell lines within a single individual. This condition arises when a mitotic nondisjunction event occurs during early embryonic development. Because this division error takes place after zygote formation, it directly generates both normal and aneuploid cell populations, explaining the mosaic state.

99. (A) The presence of two or more genetically distinct cell lines originating from a single zygote.
Genetic mosaicism refers to the occurrence of two or more distinct cell lines with different genotypes within a single individual. Crucially, all these cell lines must originate from a single fertilized egg or zygote. This distinguishes mosaicism from chimerism, which involves cell lines derived from the fusion of different zygotes.

100. (C) Mitotic nondisjunction in early cleavage.
Chromosomal mosaicism occurs when an individual possesses genetically distinct cell lines derived from a single zygote. The primary mechanism driving this condition is mitotic nondisjunction during the early cleavage stages of embryonic development. This mitotic failure divides sister chromatids unequally, creating some daughter cells with extra chromosomes and others with missing chromosomes.

101. (A) A monosomic cell line
Anaphase lag occurs during cell division when a chromosome or chromatid fails to connect to the spindle apparatus or moves too slowly toward the spindle pole. As a result, this chromosome is excluded from the forming daughter nuclei and is subsequently degraded, yielding one daughter cell that lacks that chromosome, producing a monosomic cell line.

102. (D) A localized patch of skin showing the mutated phenotype.
A mutation occurring in a somatic skin cell during early development is inherited by all subsequent daughter cells of that specific lineage. Because the mutation is restricted to this clone of cells, the animal will exhibit the mutated phenotype only in a localized, demarcated patch of skin, rather than throughout its entire body.

NODIA

103. (B) A milder phenotypic expression of the syndrome
Individuals who are mosaic for an autosomal trisomy possess both normal diploid cell lines and abnormal trisomic cell lines. Because a portion of their tissues contains genetically

normal cells, the overall gene dosage imbalance is partially mitigated. Consequently, mosaic individuals typically display a milder phenotypic expression of the syndrome compared to fully trisomic individuals.

104. (C) In a proportion of gametes but not somatic cells.
Germline mosaicism occurs when a de novo mutation arises in a primordial germ cell during early embryonic development. This mutation is subsequently propagated to a fraction of the developing gametes, while the surrounding somatic cells remain unaffected. Consequently, the individual carries the genetic mutation only in a proportion of their eggs or sperm.

105. (D) Normal parents can repeatedly produce affected offspring.
Germline mosaicism allows clinically normal parents to carry a mutation restricted entirely to their germ cells. Because their somatic cells are normal, they show no symptoms of the genetic disease. However, since a portion of their gametes carries the mutation, these unaffected parents can repeatedly produce offspring who inherit the dominant or recessive disorder.

106. (B) A subset of somatic tissues in the adult organism
Post-zygotic mutations occurring during the early blastocyst stage of embryonic development lead to somatic mosaicism. Because the mutation arises after several cell divisions have already taken place, only the descendant cells of that mutated progenitor cell will carry the genetic alteration, resulting in its distribution within a specific subset of tissues in the adult organism.

107. (A) P-3, Q-4, R-1, S-2
Epigenetic silencing of a chromosome characterizes X-inactivation, whereas a post-zygotic mutation in body tissues leads to somatic mosaicism. Germline mosaicism involves mutations isolated to gamete precursors, preventing somatic expression in the parent. Anaphase lag causes the post-zygotic loss of a chromosome, resulting in aneuploidy in a subset of descendant cellular lineages.

108. (C) Random epigenetic silencing of one X chromosome in females
The Lyon hypothesis explains dosage compensation in female mammals, where one of the two X chromosomes in each somatic cell is randomly and transcriptionally inactivated during early embryonic development. This process ensures that female cells, which have two X chromosomes, express X-linked genes at a level equivalent to hemizygous males who possess only one X chromosome.

109. (D) To equalize the dosage of X-linked gene expression between males and females
Mammalian females possess two copies of the X chromosome, while males possess only one. To prevent a potentially toxic double dosage of X-linked gene products in females, the molecular process of X-chromosome inactivation transcriptionally silences one X chromosome in female somatic cells, equalizing the functional gene expression dosage between the sexes.

110. (C) A Barr body
During interphase in female mammalian somatic cells, the inactive X chromosome remains highly condensed and heterochromatic. This structural condensation makes it visible under a light microscope as a distinct, dark-staining structure located near the inner nuclear envelope. This prominent nuclear feature is cytogenetically identified and referred to as a Barr body.

111. (B) XIST ncRNA
The X-inactive specific transcript is a non-coding RNA molecule expressed from the X-inactivation center of the chromosome destined for silencing. This specialized transcript physically coats the chromosome in cis, recruiting chromatin-modifying complexes that induce DNA methylation and histone modifications, ultimately resulting in the stable epigenetic inactivation of that specific X chromosome.

112. (D) Mosaicism driven by X-chromosome inactivation
The tortoiseshell pattern in cats results from random X-chromosome inactivation in females heterozygous for the orange and black coat color alleles. As the embryo develops, different cell lineages silence either the maternal or paternal X chromosome, creating distinct patches of skin with hair follicles expressing either orange or black pigments.

Click Here to Buy book on Amazon

113. (A) The orange/black gene is X-linked, requiring two X chromosomes for mosaicism.
The gene responsible for orange or black hair pigment is located on the X chromosome. Expressing both colors simultaneously in distinct patches requires random inactivation of different X chromosomes across somatic cell lineages. Because typical males are hemizygous with only one X chromosome, they can express only one of these pigment alleles.

114. (C) XXY
A male cat displaying a tortoiseshell coat must possess two different X chromosomes carrying the orange and black alleles, alongside a Y chromosome to determine the male phenotype. This rare genetic state is caused by chromosomal nondisjunction during gametogenesis, resulting in an XXY karyotype, which represents the feline equivalent of Klinefelter syndrome.

115. (A) The same X chromosome remains inactive in all descendant cells.
Although the initial choice of which X chromosome to inactivate is random in each embryonic cell, this decision is stably inherited through subsequent mitotic divisions. Epigenetic modifications, including DNA methylation and histone alterations, preserve the inactive state of that specific chromosome across all clonal descendant cells throughout the life of the organism.

116. (D) I and III
A chimera arises from the fusion of two or more distinct zygotes, rather than a single fertilization event. In addition, blood chimerism in twin calves is driven by placental vascular anastomoses, not meiotic nondisjunction. Conversely, tetragametic chimeras are derived from multiple zygotes, and freemartinism in heifers represents a form of hematopoietic chimerism.

117. (B) Mosaics originate from one zygote; chimeras from two or more distinct zygotes.
The primary genetic distinction between these two states lies in their developmental origins. A mosaic individual develops from a single fertilized egg that subsequent post-zygotic mutations split into genetically distinct cell lines. In contrast, a chimera is formed through the physical fusion of two or more separate zygotes early in embryonic development.

118. (C) Fusion of two independent dizygotic embryos early in gestation
A tetragametic chimera is created when two separate, independent ova are fertilized by two distinct sperm cells, forming two dizygotic embryos. If these two early-stage embryos physically fuse during the pre-implantation phase, they will develop as a single, unified organism containing cellular lineages derived from both original zygotic sources.

119. (D) Ambiguous external genitalia or true hermaphroditism
When a chimera forms from the fusion of genetically male and female embryos, the presence of both male and female sex-determining cell lineages disrupts normal sexual differentiation. This mixed genetic signal in the developing gonad and reproductive tract typically leads to the development of ambiguous external genitalia or true hermaphroditism in the animal.

120. (A) Twin pregnancies sharing placental blood vessels
Blood chimerism in domestic cattle occurs during twin gestations when the fetal membranes fuse in the uterus. This fusion establishes direct vascular connections, known as placental anastomoses, between the developing twins. These connections allow hematopoietic stem cells to pass freely between the circulations of both fetuses, establishing permanently shared blood cell populations.

NODIA

121. (B) Chimera and a freemartin
When a female calf shares a placental blood supply with a male co-twin, the exchange of fetal blood cells makes her a hematopoietic chimera. Concurrently, exposure to testicular hormones, such as anti-Müllerian hormone and testosterone, inhibits the development of her reproductive system, leading to the sterile intersex condition known as freemartinism.

122. (B) A small population of cells derived from another individual
Microchimerism is characterized by the presence of a very small, foreign population of cells that are genetically distinct from the host organism. This phenomenon is commonly established through the bidirectional transfer of fetal and maternal cells across the placental barrier during pregnancy, leaving persistent, long-lived foreign

cellular lineages within various maternal and offspring tissues.

123. (A) DNA profiling showing over two alleles at multiple loci in tissue samples
Because a tetragametic chimera is formed from the fusion of two separate zygotes, tissue samples contain cells with different genetic makeups. DNA profiling utilizing microsatellite markers or single nucleotide polymorphisms will reveal the presence of more than two distinct alleles at multiple genetic loci, confirming the contribution of more than two parental gametes.

124. (C) The hair follicles derived from two genetically distinct zygotic cell lineages
The physical appearance of patches with differing skin or hair coloration in a chimera occurs because the epidermal tissue comprises cell populations derived from two separate zygotes. If these progenitor zygotes carried different alleles for coat color, their respective cellular descendants in the skin will express distinct, visible pigmentation patterns in localized regions.

125. (A) Dizygotic twinning → Placental vascular anastomosis → Transfer of male hormones/cells → Masculinization of the female tract
The development of freemartinism begins with a dizygotic twin pregnancy. The chorionic blood vessels of the male and female fetuses fuse to create a placental vascular anastomosis. This physical connection allows the systemic transfer of male hormones and hematopoietic cells into the female twin, actively suppressing her female tract and inducing partial masculinization.

126. (C) Sterile female calf born co-twin to a male
A freemartin is defined as an infertile female calf born as a twin to a male calf. This condition arises due to intrauterine exposure to male hormones transmitted through shared placental circulation. These hormones permanently disrupt the normal development of the female reproductive tract, rendering the heifer sterile while the male co-twin remains fertile.

127. (B) Hematopoietic cells and Anti-Müllerian hormone
During gestation, placental vascular connections facilitate the exchange of blood between male and female bovine twins. The systemic transfer of male anti-Müllerian hormone actively regression of the female's Müllerian ducts, while the simultaneous migration of male hematopoietic stem cells establishes long-term cellular chimerism within the bone marrow and immune system of the female twin.

128. (D) Hypoplastic ovaries, underdeveloped uterus, and seminal vesicles
The reproductive tract of a freemartin heifer is severely hypoplastic and masculinized. Because anti-Müllerian hormone from the male co-twin suppresses normal female development, the ovaries remain small and non-functional, the uterus is rudimentary or absent, and the anterior vagina is shortened. In contrast, male accessory glands like seminal vesicles may partially develop.

129. (A) The presence of both XX and XY hematopoietic cells
Hematopoietic chimerism in a freemartin calf is identified by the coexistence of two genetically distinct blood cell populations. Due to the sharing of fetal circulation with her male twin, the female calf acquires and permanently hosts hematopoietic stem cells from the male, resulting in a mixture of both XX and XY cells in her blood.

130. (C) Placental vascular fusions occur very rarely in sheep and pigs.
Although twins and large litters are common in sheep and pigs, freemartinism is rare in these species. This is because the fetal membranes of adjacent embryos in the uterus do not typically form vascular connections. Without these placental anastomoses, there is no exchange of blood, preventing the transfer of masculinizing hormones to female fetuses.

131. (A) PCR detection of Y-chromosome sequences in a blood sample
The most definitive diagnostic method to confirm freemartinism is a polymerase chain reaction test performed on a blood sample. Since the freemartin heifer is a hematopoietic chimera due to cell sharing with her male co-twin, her blood will contain male cells. Detecting Y-chromosome specific gene sequences confirms the presence of male DNA.

132. (D) Usually fertile, but may have reduced semen quality.
The male co-twin of a freemartin heifer is generally fertile and develops normal male reproductive anatomy. However, because he also

Downloaded from www.pdf.tube

excess chromosomes. If an unreduced polar body is retained, the egg remains diploid rather than haploid. When fertilized by a normal haploid sperm, the zygote receives three complete sets of chromosomes, resulting in a triploid organism.

52. (C) Odd chromosome sets cannot pair properly in meiosis, yielding unbalanced gametes.
Triploid organisms carry an odd number of chromosome sets, which prevents homologous chromosomes from pairing uniformly during prophase I of meiosis. Consequently, chromosome segregation is highly irregular and chaotic, producing unbalanced, aneuploid gametes. These genetically defective gametes are nonviable, resulting in complete physiological sterility of the triploid individual.

53. (C) A is true but R is false.
Triploidy is occasionally observed in live-born chickens and other poultry, presenting with characteristic developmental and phenotypic abnormalities. However, this is not because avian species tolerate these genetic imbalances better due to microchromosomes. In fact, large chromosomal duplications are highly deleterious across birds, making the reasoning behind this assertion false.

54. (D) Interstitial deletion
An interstitial deletion is a structural chromosome mutation that requires two double-strand breaks along a single chromosome arm. The segment located between the two break points is lost, and the remaining ends are joined back together. This differs from a terminal deletion, which involves only one break near the chromosome end.

55. (B) Paracentric inversion
A paracentric inversion occurs when a chromosomal segment undergoes two breaks, rotates one hundred and eighty degrees, and is reinserted back into the chromosome without including the centromere. Because both break points lie within a single chromosome arm, the relative lengths of the short and long arms remain completely unchanged.

56. (C) Production of dicentric and acentric chromatids.
Recombination within a paracentric inversion loop during meiosis produces unbalanced chromatids. Specifically, a single crossover event inside the loop generates a dicentric chromatid with two centromeres and an acentric fragment with no centromere. The acentric fragment is lost, and the dicentric chromatid breaks during anaphase, resulting in nonviable gametes.

57. (D) Provide new genetic material for the evolution of gene families.
Gene duplications supply extra copies of functional genes to the genome. While one gene copy maintains its original essential biological function, the duplicated copy is freed from selective constraints. Over evolutionary time, this redundant copy can accumulate mutations and acquire novel functions, driving the development of diverse gene families.

58. (A) Pseudodominance
When a chromosome undergoes a large deletion, the matching homologous chromosome retains its alleles. If the remaining chromosome carries a recessive allele in the deleted region, that recessive phenotype will be expressed because there is no dominant allele to mask it. This phenotypic expression of a recessive allele is termed pseudodominance.

59. (B) Pericentric inversions may alter the chromosome arm ratio.
Pericentric inversions involve two break points located on opposite sides of the centromere. When the segment rotates and reattaches, the centromere position often shifts, changing the relative lengths of the p and q arms. Paracentric inversions occur on only one arm, keeping the centromere position and arm ratio completely unchanged.

NODIA

60. (B) A partial short arm deletion
Cri-du-chat syndrome is a well-known genetic disorder caused by a structural chromosome deletion. Specifically, it involves a partial deletion of the short arm of chromosome five. Similar congenital syndromes and developmental defects in domestic animals are caused by analogous heterozygous partial deletions of specific chromosomal short arms in their genomes.

61. (C) Chromosomal insertion
A chromosomal insertion is a complex structural rearrangement that requires three distinct double-strand breaks. Two breaks are necessary to liberate a segment of DNA from a donor chromosome, while a third break is required on a recipient chromosome to open up the insertion

142. (C) 66.7%
Mating two carrier animals produces a genotypic ratio of one homozygous normal, two heterozygous carriers, and one homozygous recessive. Because the homozygous recessive condition is prenatally lethal, these embryos are aborted. Out of the remaining viable, liveborn offspring, two-thirds, or approximately sixty-six point seven percent, are expected to be heterozygous carriers of the mutation.

143. (A) Both (A) and (R) are true and (R) is the correct explanation of (A).
Deficiency of uridine monophosphate synthase is maintained at low frequencies in dairy cattle because homozygous embryos do not survive gestation. These embryos fail to synthesize pyrimidine nucleotides de novo, which are vital for DNA replication and RNA synthesis. This biochemical block results in embryonic death and pregnancy loss around day forty of gestation.

144. (D) Pyrimidine biosynthesis pathway
Deficiency of uridine monophosphate synthase is an inherited metabolic defect that disrupts the de novo pyrimidine biosynthesis pathway. The affected enzyme is responsible for catalyzing the final steps of this pathway, which converts orotic acid to uridine monophosphate. This product is a vital precursor for all pyrimidine nucleotides required for cellular division.

145. (A) Appears normal but excretes high levels of orotic acid
A dairy cow that is a heterozygous carrier for the uridine monophosphate synthase mutation displays a completely normal physical appearance and production performance. However, because she possesses only fifty percent of normal enzyme activity, she cannot fully process pathway intermediates, leading to the metabolic accumulation and urinary excretion of high levels of orotic acid.

146. (C) Urea cycle
Citrullinemia is an autosomal recessive metabolic disorder that directly disrupts the urea cycle in the liver. This essential biochemical pathway is responsible for converting toxic ammonia, a byproduct of protein catabolism, into water-soluble urea for excretion. When this cycle is blocked, ammonia accumulates in tissues, leading to acute systemic toxicity.

147. (A) Argininosuccinate synthetase (ASS)
Bovine citrullinemia is caused by a single point mutation that creates a premature stop codon in the gene encoding argininosuccinate synthetase. This nonsense mutation results in a completely non-functional enzyme. Consequently, the liver cannot catalyze the conversion of citrulline and aspartate into argininosuccinate, which completely halts the urea cycle.

148. (D) Ammonia
The structural failure of the enzyme argininosuccinate synthetase blocks the urea cycle, preventing the metabolic conversion of nitrogenous waste. This biochemical block causes a rapid, life-threatening accumulation of ammonia in the blood of affected calves. High ammonia levels cross the blood-brain barrier, causing severe, irreversible swelling and damage to central nervous tissues.

149. (B) Progressive neurological deficits ending in death
Calves homozygous for the citrullinemia mutation appear normal at birth but develop severe clinical symptoms within their first week. As toxic ammonia accumulates in the brain, they exhibit progressive neurological deficits, including depression, head pressing, blindness, convulsions, and coma, rapidly culminating in death if supportive clinical therapy is not administered.

150. (C) Unexplained abortions and early calf mortality
Recessive genetic disorders like deficiency of uridine monophosphate synthase and citrullinemia result in substantial economic losses within the dairy industry. These financial impacts stem primarily from reproductive failures, including early embryonic loss and unexplained abortions, as well as the inevitable mortality of affected calves shortly after birth, reducing herd replacement rates.

151. (D) Molecular tests can accurately identify healthy, asymptomatic carriers.
While biochemical assays only detect abnormalities in affected animals with active symptoms, molecular assays analyze DNA directly. This allows molecular testing to identify healthy, asymptomatic heterozygous carriers that possess one copy of the mutant allele. Identifying these carriers is crucial for breeding management

Click Here to Buy book on Flipkart

insemination sires. Screening these influential animals prior to semen distribution prevents the widespread dissemination of deleterious recessive alleles and protects the genetic health of the global dairy population.

****

NODIA

161. (A) I and III
A fully lethal gene causes death before reproductive age, while a semi-lethal gene causes the death of more than fifty percent but less than one hundred percent of affected individuals. Sub-lethal genes cause death before sexual maturity but after birth, not strictly during the early embryonic stage of development.

162. (C) An allele that causes the death of the organism, invariably reducing its evolutionary fitness to zero
In population genetics, a fully lethal allele is defined as one that causes the death of the organism before it can reach reproductive maturity. Because the affected individual cannot produce offspring to pass on its genetic material, its evolutionary fitness is reduced to zero, preventing direct transmission of that homozygous genotype.

163. (A) Affected individuals die before they can reproduce.
Dominant lethal alleles are rarely transmitted through generations because they exert their fatal effects in both the heterozygous and homozygous states. Affected individuals die or fail to develop before reaching reproductive age, preventing them from producing offspring and passing the mutation on to subsequent generations, unless the lethality has a delayed onset.

164. (D) Sub-lethal
An allele that causes the death of less than fifty percent of affected individuals before they reach reproductive age is classified as sub-lethal. Because eighty percent of the affected calves survive to maturity and reproduce, the mortality rate is low, distinguishing this mutation from more severe semi-lethal or fully lethal genetic defects.

165. (B) Normal heterozygous carriers who reproduce successfully
Recessive lethal alleles persist in populations because they do not affect the survival or reproduction of heterozygous individuals. These carriers carry one copy of the mutation without exhibiting clinical signs. They reproduce normally, unknowingly passing the recessive allele to fifty percent of their offspring, thereby maintaining the mutation within the gene pool.

166. (C) Conditional lethal mutation
A conditional lethal mutation is an alteration that is compatible with survival under permissive environmental conditions but causes death under specific, non-permisive conditions. These environmental triggers can include temperature changes, dietary components, or exposure to specific chemicals, demonstrating how external environmental factors directly determine the lethality of certain genetic alleles.

167. (D) Rendering gametes non-viable or unable to fertilize
Gametic lethal mutations exert their effects within the haploid stage of the life cycle. Instead of causing embryonic death, these mutations compromise the viability, motility, or functional capacity of the sperm or egg cells. This impairment prevents fertilization, meaning the affected alleles are not transmitted to form a diploid zygote.

NODIA

168. (A) Overdominance with recessive lethality
This genetic scenario demonstrates overdominance combined with recessive lethality, where heterozygous individuals exhibit a unique, viable phenotype that is favored or selected by breeders. However, because the allele is lethal in the homozygous state, mating these heterozygotes results in a loss of homozygous offspring, which fail to develop or survive.

169. (B) Severe achondroplasia, shortened limbs, and facial dysmorphia
The "Bulldog calf" anomaly in Dexter cattle represents a severe form of chondrodysplasia. Affected calves are born aborted or dead at term, presenting with a highly characteristic phenotype that includes extreme achondroplasia, severely shortened limbs, a compressed vertebral column, a protruding tongue, and pronounced facial dysmorphia resembling a bulldog.

170. (B) 2:1:1
Mating two short-legged Dexter cattle, which are heterozygous carriers, yields a genotypic ratio of one homozygous normal, two heterozygous short-legged, and one homozygous recessive bulldog calf. This translates to a phenotypic ratio at birth or conception of two short-legged animals to one normal long-legged Kerry-type animal, and one severely deformed bulldog calf.

to prevent carrier-to-carrier matings that produce diseased offspring.

152. (A) P-3, Q-4, R-2, S-1
Bovine leukocyte adhesion deficiency is caused by a mutation in the CD18 gene, while citrullinemia is linked to a deficiency in argininosuccinate synthetase. Deficiency of uridine monophosphate synthase is caused by a mutant UMP synthase enzyme, and complex vertebral malformation arises from a point mutation within the SLC35A3 gene.

153. (B) Multiple additive genes, often influenced by environment
Polygenic inheritance is characterized by traits that are determined by the cumulative action of multiple independent genes, each exerting a small, additive effect on the phenotype. Unlike monogenic traits, these quantitative characteristics present a continuous spectrum of variation and are highly susceptible to modification by environmental factors such as nutrition and management.

154. (B) Interaction of multiple loci with diet and exercise
Canine hip dysplasia is a classic example of a multifactorial trait. Its phenotypic expression is not governed by a single mutation but rather by the complex interaction of multiple genetic loci with non-genetic environmental factors, such as rapid growth rates, high caloric intake, and excessive physical joint stress during early developmental skeletal phases.

155. (A) A DNA region associated with a continuously varying trait
A quantitative trait locus is a specific section of DNA that is statistically associated with variation in a complex, continuously distributed phenotype. These genomic regions contain one or more genes that contribute to the expression of polygenic traits, such as growth rate, milk yield, or disease susceptibility, rather than determining a simple binary state.

156. (C) The proportion of phenotypic variance in a population attributable to genetic variance
In quantitative genetics, herizability measures the proportion of total phenotypic variation observed within a specific population that is due to additive genetic differences among individuals. This population-level statistic indicates how effectively phenotypic selection can alter a trait, such as canine hip dysplasia, but does not dictate the genetic inheritance of an individual dog.

157. (D) Estimating breeding values based on phenotypic evaluations (e.g., radiograph scores) of the individual and its relatives
Because polygenic disorders are controlled by numerous loci, simple DNA tests cannot identify carriers. The most effective strategy to reduce disease incidence is to estimate breeding values using phenotypic data, such as hip radiograph scores, collected from the individual and its close relatives. Breeders can then select animals with superior genetic merit.

158. (C) Its combined genetic liability and environmental exposure exceed a critical threshold point.
The threshold model describes traits that present biochemically as binary conditions but are determined by an underlying, continuous genetic liability. An animal displays the clinical phenotype when its cumulative genetic susceptibility, combined with predisposing environmental influences, surpasses a specific, critical threshold value, causing the disease state to manifest outwardly.

159. (B) The exact combination of contributing alleles is usually unknown, and environmental factors mask the underlying genotype.
Eradicating polygenic disorders is challenging because they are governed by multiple genes with small individual effects, and the precise causative alleles are often unidentified. Furthermore, environmental variations can alter or mask the genetic potential, making it difficult to accurately assess an animal's true breeding value based solely on its outward phenotype.

160. (D) Gene-environment interaction
Reducing caloric intake in dogs genetically predisposed to hip dysplasia clearly demonstrates a gene-environment interaction. This fundamental genetic concept highlights how the final physical phenotype is shaped by environmental modifications acting upon an underlying genotype, showing that environmental management can significantly mitigate the clinical severity of a hereditary orthopedic condition.

Downloaded from www.pdf.tube

notably prevalent within the Arabian horse breed. Affected foals carry two copies of the mutated gene and cannot produce functional adaptive immune cells, leaving them completely vulnerable to various opportunistic infections that invariably lead to death within the first few months.

182. (C) They fail to produce functional B and T lymphocytes.
At birth, foals with severe combined immunodeficiency are temporarily protected by maternal antibodies absorbed from colostrum. However, as these maternal antibodies naturally decline, the foal's immune system cannot defend itself because the genetic mutation prevents the development of functional B and T lymphocytes, which are essential for mounting an adaptive immune response.

183. (D) DNA-dependent protein kinase catalytic subunit
The molecular defect responsible for severe combined immunodeficiency in Arabian horses is a five-base-pair deletion within the gene that encodes the catalytic subunit of DNA-dependent protein kinase. This deletion shifts the reading frame and introduces a premature stop codon, resulting in a non-functional enzyme that cannot repair DNA double-strand breaks.

184. (A) V(D)J recombination of antigen receptor genes
The mutation in the DNA-dependent protein kinase catalytic subunit directly impairs the process of V(D)J recombination. This pathway is essential for rearranging the gene segments that encode immunoglobulin and T-cell receptor proteins. Without this DNA repair mechanism, developing lymphocytes cannot generate diverse antigen receptors, halting their normal cellular maturation.

185. (B) Hirschsprung's Disease
Overo lethal white syndrome in horses serves as a clinical model for Hirschsprung's disease in humans. Both genetic conditions are characterized by a congenital absence of intrinsic ganglion cells in the distal gastrointestinal tract, resulting from defective neural crest cell migration. This lack of innervation leads to functional intestinal obstruction and megacolon.

186. (B) Autosomal recessive
Because heterozygous carriers of the severe combined immunodeficiency mutation show absolutely no clinical abnormalities and are phenotypically indistinguishable from non-carriers, the trait is classified as autosomal recessive. The disease only manifests when an offspring inherits two copies of the mutated gene, one from each of its heterozygous carrier parents.

187. (A) Severe skeletal deformities, elongated limbs, and spinal curvature
Spider lamb syndrome is an inherited chondrodysplasia that primarily affects Suffolk and Hampshire sheep. The condition is characterized by severe skeletal deformities, including abnormally long, bent, and fine-boned limbs, scoliosis, kyphosis, and facial deviations. These skeletal abnormalities prevent affected lambs from standing or walking normally, leading to early mortality.

188. (A) Both (A) and (R) are true and (R) is the correct explanation of (A).
Porcine stress syndrome can cause sudden death in pigs during handling or transport. This susceptibility is due to a point mutation in the RYR1 gene, which alters the calcium release channel in skeletal muscle. Under stress or exposure to halothane, the channel remains open, leading to hypermetabolism, malignant hyperthermia, and fatal muscle spasms.

NODIA

189. (D) Calcium release channel in skeletal muscle sarcoplasmic reticulum
The ryanodine receptor gene mutated in porcine stress syndrome encodes a calcium release channel located in the sarcoplasmic reticulum membrane of skeletal muscle cells. This channel is crucial for excitation-contraction coupling, regulating the release of calcium ions into the sarcoplasm to initiate muscle contraction in response to motor nerve signals.

190. (C) Carriers produced highly muscled, lean carcasses of high economic value.
Before molecular testing was available, swine breeders selected for the RYR1 mutation because heterozygous carrier pigs exhibited extreme muscling, rapid growth, and minimal carcass fat. This highly desirable commercial conformation provided a significant economic advantage at market, which led to the widespread

2015

8. Cerebellar hypoplasia in feline panleukopenia virus infection is a developmental failure primarily caused by which mechanism?
(A) Viral destruction of mitotic cells during organogenesis
(B) Severe autoimmune-mediated attack on mature Purkinje cells
(C) Complete ischemic necrosis of the major cerebellar peduncles
(D) Inherited genetic mutation in the host nuclear DNA structure

2006

9. Which of the following is an example of localized tissue hypoplasia?
(A) Diffuse enlargement of the follicular thyroid gland
(B) Segmental underdevelopment of the intestine
(C) Physiological involution of the postpartum uterus
(D) Chronic wasting of skeletal muscle from malnutrition

2009

10. Analyze the assertion and reason statements below to select the correct option.
Assertion (A): Disuse atrophy is rapidly reversible once normal physical activity is resumed.
Reason (R): Atrophic cells are dead and must be replaced by tissue stem cells during recovery.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2003

11. Which cellular mechanism primarily mediates the degradation of intracellular proteins during atrophy?
(A) The ubiquitin-proteasome pathway
(B) The caspase-dependent apoptotic cascade
(C) The cytochrome c release pathway
(D) The telomerase activation mechanism

2006

12. Denervation atrophy of skeletal muscle typically follows damage to which anatomical structure?
(A) The upper motor neuron in the cerebral cortex
(B) The neuromuscular junction receptors
(C) The lower motor neuron or its peripheral axon
(D) The sensory nerve fibers supplying the muscle

2007

13. Which condition is a direct cause of ischemic atrophy?
(A) Pathological overproduction of active growth hormone
(B) Complete, sudden occlusion of a primary major artery
(C) Chronic autoimmune destruction of an endocrine gland
(D) Gradual reduction of arterial blood supply

2013

14. Senile atrophy in the brain is characterized morphologically by which feature?
(A) Narrowing of sulci and widening of gyri
(B) Massive proliferation of microglial cells
(C) Widening of sulci and narrowing of gyri
(D) Rapid accumulation of intracytoplasmic lipid

2010

15. Consider the following statements regarding endocrine atrophy:
I. It occurs when a target organ loses its hormonal stimulation.
II. Loss of ACTH leads to hypertrophy of the adrenal cortex.
III. Castration leads to atrophy of the prostate gland.
Which of the above statements is/are correct?
(A) I and II
(B) II and III
(C) I and III
(D) I only

2019

16. What is the defining characteristic of pathologic hyperplasia compared to neoplasia?
(A) Pathologic hyperplasia is irreversible upon stimulus removal
(B) Pathologic hyperplasia involves monoclonal cell proliferation
(C) Pathologic hyperplasia occurs exclusively in epithelial tissues
(D) Pathologic hyperplasia ceases when the stimulus is removed

2005

17. Cystic endometrial hyperplasia in canines is most commonly driven by the excessive influence of which hormone?
(A) Progesterone
(B) Testosterone
(C) Somatostatin

Click Here to Buy book on Amazon

proceed through multiple cycles without manual replenishment.

201. (B) SNP Microarray
SNP microarrays utilize hybridization technology, where fluorescently labeled single-stranded target DNA prepared from a sample is hybridized to complementary oligonucleotide probes immobilized on a solid surface. This method allows the simultaneous interrogation of hundreds of thousands of single nucleotide polymorphisms, providing a rapid and cost-effective tool for genomic selection.

202. (B) PCR identifies carriers at birth, avoiding costly test matings.
PCR-based genotyping offers a rapid, highly accurate, and non-invasive alternative to traditional progeny testing. Instead of conducting time-consuming and expensive test matings to monitor offspring for recessive phenotypes, DNA can be extracted from hair or blood at birth, allowing immediate carrier identification and accelerating genetic progress in breeding programs.

203. (A) Heterozygous
A carrier is defined as an individual that is heterozygous at a specific genetic locus, possessing one normal wild-type allele and one mutant recessive allele. Because the normal allele is fully dominant, the carrier remains phenotypically healthy and free of disease, but can pass the mutant allele to fifty percent of its offspring.

204. (C) Male
In clinical genetics and pedigree analysis, standard symbol conventions are used to represent family structures and genetic inheritance. A square symbol represents a male individual, while a circle represents a female. Shaded symbols denote affected individuals, and half-shaded or dotted symbols are used to identify confirmed heterozygous carriers of the trait.

205. (D) Systematically identify and manage carrier animals in breeding
The primary objective of a genetic eradication strategy is to systematically identify and manage carrier animals within a breeding population. By utilizing molecular testing to identify asymptomatic heterozygous individuals, breeders can design strategic matings that avoid crossing two carriers, preventing the birth of homozygous affected animals and gradually reducing the mutant allele frequency.

206. (C) I, III and IV
Molecular assays provide accurate carrier identification, and pedigree analysis can estimate carrier probabilities based on ancestral records. Rigorous testing of artificial insemination sires is critical due to their extensive impact on allele frequencies. However, immediately culling all carriers is often economically impractical and damages genetic diversity, while genomic selection does account for recessive mutations.

207. (B) It reduces genetic diversity, increasing inbreeding and other defects.
Culling all identified carriers can be highly detrimental, particularly in small populations. This aggressive approach drastically reduces the size of the breeding pool, leading to a loss of overall genetic diversity and increasing the rate of inbreeding. Consequently, this can expose other recessive defects and compromise the selection potential for economically important traits.

208. (D) Mating carriers to clear animals and selecting only clear offspring
To preserve valuable genetic lines without producing affected animals, breeders can mate confirmed heterozygous carriers to individuals tested clear of the mutation. The resulting offspring must be genotyped, and only those that are clear are selected for future breeding, allowing the gradual elimination of the mutant allele while retaining beneficial genetics.

209. (A) Mate him to his own daughters or carrier females and observe progeny
When molecular tests are unavailable, breeders rely on test matings to detect recessive carrier bulls. Mating the suspect sire to his own daughters, or to known carrier females, exposes recessive alleles. If no affected offspring are produced from a statistically sufficient number of progeny, the bull is declared clear with high confidence.

NODIA

210. (C) Mandatory genetic screening of elite AI sires before semen distribution
The rapid global spread of recessive defects like bovine leukocyte adhesion deficiency and complex vertebral malformation highlights the necessity of mandatory genetic screening for elite artificial

dissemination of the deleterious allele throughout the breeding population.

191. (B) Bone growth via inhibition of chondrocytes
The FGFR3 gene encodes fibroblast growth factor receptor 3, which is a key regulator of endochondral ossification. Under normal physiological conditions, activation of this receptor inhibits the proliferation and differentiation of chondrocytes in the growth plates of long bones, thereby controlling the rate and extent of longitudinal bone growth.

192. (D) Porcine Stress Syndrome (PSS)
Malignant hyperthermia is the clinical manifestation of the RYR1 gene mutation when triggered by volatile anesthetics, such as halothane. This condition is physiologically identical to the crisis seen in porcine stress syndrome, where mutated calcium release channels cause an uncontrolled rise in core body temperature, muscle rigidity, and metabolic acidosis.

193. (A) It becomes pale, soft, and exudative (PSE).
Pigs that undergo a stress crisis before slaughter produce meat classified as pale, soft, and exudative. The rapid post-mortem breakdown of glycogen under high body temperatures and acidic conditions denatures muscle proteins. This denaturation reduces the water-holding capacity of the muscle, resulting in a soft texture and poor meat quality.

194. (C) The T to A FGFR3 gene mutation using PCR
To eliminate spider lamb syndrome from a breeding flock, breeders utilize a PCR-based DNA test. This assay directly identifies the causative T to A point mutation in the FGFR3 gene. By analyzing DNA from blood or tissue samples, this test accurately distinguishes between homozygous normal, heterozygous carrier, and homozygous affected animals.

195. (A) To exponentially amplify target DNA
The primary function of the polymerase chain reaction is to amplify a specific target DNA sequence from a complex genomic template. Through repeated cycles of denaturation, primer annealing, and enzymatic extension, PCR generates millions of copies of the desired sequence, providing sufficient genetic material for subsequent diagnostic analysis or sequencing.

196. (D) By using restriction enzymes that cut only one of the alleles
Restriction fragment length polymorphism distinguishes between alleles by utilizing restriction endonucleases that recognize specific, palindromic DNA sequences. A point mutation can create or destroy one of these recognition sites. Consequently, digesting the PCR product yields fragments of different sizes, which are then separated and visualized using gel electrophoresis.

197. (B) P-2, Q-4, R-3, S-1
Polymerase chain reaction is used to amplify specific DNA segments, whereas restriction fragment length polymorphism differentiates alleles based on restriction enzyme cleavage. Single nucleotide polymorphism arrays allow the simultaneous genotyping of thousands of markers, and cytogenetic karyotyping remains the standard method to visualize and detect large structural chromosomal rearrangements.

198. (C) Simultaneous genotyping of thousands of markers across the genome
Single nucleotide polymorphism arrays are highly valuable in livestock breeding because they allow the high-throughput genotyping of thousands of genetic markers distributed across the entire genome. This comprehensive genetic profiling supports genomic selection, the identification of quantitative trait loci, and the calculation of highly accurate genomic breeding values for complex traits.

199. (D) Bands from both the normal and mutant allele digests
A heterozygous carrier bull possesses one normal allele and one mutant allele. During PCR-RFLP analysis, the restriction enzyme will cleave only the allele containing the specific restriction site. Consequently, gel electrophoresis of the digested DNA will display a combination of bands representing both the cleaved mutant fragments and the intact normal fragments.

200. (A) It remains stable and active at high denaturation temperatures.
Taq polymerase is a thermostable DNA polymerase isolated from the thermophilic bacterium Thermus aquaticus. This enzyme is essential for the polymerase chain reaction because it can withstand the high denaturation temperatures required to separate DNA strands without losing activity, allowing the reaction to

Downloaded from www.pdf.tube

CHAPTER 06
Disturbances in Cell Growth and Oncology

2007
1. Which of the following defines cellular hypertrophy?
(A) An increase in cell size leading to an increase in organ size
(B) An increase in cell numbers leading to increased organ volume
(C) A reversible replacement of one adult cell type by another type
(D) A reduction in organ size from decreased cell size and number

2010
2. Which fundamental mechanism primarily drives physiological hypertrophy in striated muscle cells?
(A) Rapidly increased rate of mitotic cellular division
(B) Intracellular accumulation of neutral lipid droplets
(C) Increased synthesis of proteins and organelles
(D) Direct transformation of fibroblasts into myocytes

2021
3. Hyperplasia is a cellular adaptation that can only occur in which type of tissue?
(A) Tissues containing cell populations capable of replication
(B) Tissues composed of permanently post-mitotic cells
(C) Tissues capable of synthesizing large amounts of collagen
(D) Tissues devoid of a blood supply

2022
4. Which of the following represents a classic example of compensatory hyperplasia?
(A) Enlargement of the left ventricle in systemic hypertension
(B) Hypertrophy of skeletal muscle during weightlifting
(C) Physiological shrinkage of the thymus during puberty
(D) Regeneration of liver tissue after partial hepatectomy

2010
5. Match the growth failures in List I with their corresponding definitions in List II.
List I: (Growth Failures)
P. Agenesis
Q. Aplasia
R. Hypoplasia
S. Atrophy
List II: (Definitions)
1. Failure of an organ to develop beyond a rudimentary primordium
2. Complete lack of formation of an organ and its primordium
3. Shrinkage of a previously normally developed organ
4. Underdevelopment of an organ with fewer than normal cells
(A) P-2, Q-1, R-4, S-3
(B) P-1, Q-2, R-3, S-4
(C) P-3, Q-4, R-1, S-2
(D) P-4, Q-3, R-2, S-1

2007
6. Unilateral renal agenesis is best described by which of the following statements?
(A) The kidney is physically present but microscopically rudimentary
(B) The kidney formed normally but subsequently shrank due to ischemia
(C) The kidney failed to form due to absence of the ureteric bud
(D) The kidney is poorly developed and cannot support normal filtration

NODIA

2021
7. What is the distinguishing morphological feature of aplasia compared to agenesis?
(A) Agenesis always leaves a fibrous scar, while aplasia leaves a fluid-filled cyst
(B) Aplasia only affects paired organs, whereas agenesis affects singular organs
(C) Agenesis involves hyperplastic cells, whereas aplasia involves apoptotic cells
(D) Aplasia has a rudimentary tissue mass, whereas agenesis lacks a primordium

171. (A) Recessive lethal trait with incomplete dominance
The Dexter dwarfism trait is inherited as an autosomal recessive lethal condition with incomplete dominance. Heterozygous animals carry one copy of the mutated gene and express a mild, short-legged phenotype that is viable. In contrast, homozygous recessive individuals receive two copies of the mutant allele, resulting in the lethal bulldog calf phenotype.

172. (C) ACAN (Aggrecan)
The mutation responsible for Dexter achondroplasia and the associated bulldog calf phenotype is located within the ACAN gene, which encodes the proteoglycan aggrecan. Aggrecan is a critical structural component of the extracellular matrix in cartilage, and its disruption prevents the normal development and mineralization of the skeletal system during gestation.

173. (D) Endochondral ossification process
The aggrecan mutation in Dexter cattle directly impairs the process of endochondral ossification, which is the vital pathway where cartilage is replaced by bone during embryonic development. Without functional aggrecan proteoglycans, chondrocytes fail to organize and mature properly, halting the longitudinal growth of long bones and leading to severe skeletal defects.

174. (C) Mating a short-legged Dexter to another short-legged Dexter
Because the short-legged phenotype in Dexter cattle is determined by a heterozygous genotype, mating two short-legged individuals carries a twenty-five percent risk of producing a homozygous recessive offspring. This mating will result in the birth of a severely malformed and lethal bulldog calf, which breeders must avoid to maintain herd welfare.

175. (B) They exhibit a normal, long-legged Kerry-type phenotype.
Homozygous normal individuals in the Dexter breed carry two copies of the wild-type aggrecan allele and do not exhibit any form of achondroplasia. Consequently, these animals develop with a normal, long-legged stature that is phenotypically indistinguishable from Kerry cattle, and they are completely free from the risk of producing bulldog calves.

176. (D) Achondroplasia
The genetic inheritance and clinical manifestations of the Dexter cattle dwarfism mutation share strong parallels with human achondroplasia. In humans, heterozygous mutations in the FGFR3 gene cause a viable short-limbed dwarfism, while inheriting two copies of the mutated allele is homozygous lethal, leading to severe skeletal defects and neonatal death.

177. (A) Fetal development, causing abortion around 7 months
The lethal nature of the homozygous aggrecan mutation in Dexter cattle typically manifests during late fetal development. The severe skeletal deformities and internal fluid accumulations associated with this bulldog phenotype lead to fetal death and spontaneous abortion, commonly occurring around seven months of gestation, rather than during postnatal life.

178. (C) Frame overo pinto pattern
Overo lethal white syndrome is a genetic disorder associated with the frame overo coat color pattern in horses. Heterozygous horses carry one copy of the mutation and display a desirable spotted coat. However, breeding two frame overos can produce homozygous white offspring that inherit the lethal physiological defects associated with this syndrome.

179. (A) I, II and III
Overo lethal white syndrome is caused by a dinucleotide mutation in the EDNRB gene. Affected homozygous foals are born completely white and suffer from intestinal aganglionosis, which prevents normal bowel movement. The condition is uniformly fatal and cannot be cured by surgical intervention, making prompt euthanasia the only humane option.

180. (D) Intestinal impaction from absent functional nerve ganglia in bowel
Foals afflicted with overo lethal white syndrome fail to develop functional nerve ganglia in the ileum and colon due to a defect in neural crest cell migration. This lack of innervation prevents intestinal peristalsis, leading to severe colic and painful intestinal impaction, which is the primary cause of death in these animals.

181. (B) Arabian
Severe combined immunodeficiency is an autosomal recessive lethal disorder that is

Click Here to Buy book on Flipkart

(D) Gonadotropin

2006

18. Goiter is an example of pathologic hyperplasia occurring in which organ?
(A) Adrenal gland
(B) Parathyroid gland
(C) Thyroid gland
(D) Pituitary gland

2007

19. A deficiency of which dietary element is classically associated with the development of hyperplastic goiter?
(A) Iodine
(B) Calcium
(C) Iron
(D) Copper

2010

20. Select the correct sequence of cellular events leading to compensatory hyperplasia in the liver.
(A) Cell cycle entry → Priming by cytokines → Growth factor stimulation → Proliferation
(B) Priming by cytokines → Growth factor stimulation → Cell cycle entry → Proliferation
(C) Growth factor stimulation → Cell cycle entry → Priming by cytokines → Proliferation
(D) Proliferation → Cell cycle entry → Growth factor stimulation → Priming by cytokines

2006

21. Which statement best differentiates metaplasia from dysplasia?
(A) Metaplasia represents a permanent malignant transformation, while dysplasia is a benign process
(B) Metaplasia is a change to another mature cell type, while dysplasia is disordered cell growth
(C) Metaplasia occurs only within connective tissues, while dysplasia occurs only in epithelium
(D) Metaplasia involves cellular atrophy, while dysplasia involves progressive cellular hypertrophy

2014

22. Squamous metaplasia in the respiratory tract of avians is highly indicative of a deficiency in which vitamin?
(A) Vitamin C
(B) Vitamin D
(C) Vitamin A
(D) Vitamin K

2008

23. What is the fundamental cellular reprogramming mechanism that causes metaplasia?
(A) Direct transdifferentiation of one mature cell type into another mature cell type
(B) Fusion of two distinct mature somatic cell types to create a hybrid cell lineage
(C) Viral integration of foreign DNA into the genome of mature epithelial cells
(D) Reprogramming of stem cells to differentiate along a new mature pathway

2016

24. Which feature is an essential morphological hallmark of epithelial dysplasia?
(A) Loss of architectural orientation and cell uniformity
(B) Consistent maintenance of uniform cellular size and shape
(C) Pronounced decrease in the nuclear-to-cytoplasmic ratio
(D) Complete absence of mitotic figures in the tissue layers

2021

25. Match the specific metaplastic changes in List I with their typical inducing conditions in List II.
List I: (Metaplastic Changes)
P. Squamous metaplasia of respiratory epithelium
Q. Osseous metaplasia in connective tissue
R. Squamous metaplasia of esophageal glands
S. Myeloid metaplasia (extramedullary hematopoiesis)
List II: (Inducing Conditions)
1. Chronic irritation by cigarette smoke or chronic inflammation
2. Chronic trauma or old scars in soft tissue
3. Vitamin A deficiency in parrots
4. Severe chronic anemia disrupting bone marrow function
(A) P-1, Q-2, R-3, S-4
(B) P-2, Q-3, R-1, S-4
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-2, S-3

2024

26. Which scenario describes a mesenchymal metaplasia?
(A) Transformation of ciliated epithelium to squamous epithelium
(B) Formation of bone within an area of chronic muscle injury
(C) Replacement of squamous epithelium by columnar epithelium

Downloaded from www.pdf.tube

normally made by the tissue
(B) Decreased reliance on glycolysis for energy
(C) Elaboration of fetal proteins such as α-fetoprotein
(D) Enhanced contact inhibition

2005
47. Chemical carcinogenesis is broadly divided into two major stages known as:
(A) Invocation and progression
(B) Initiation and promotion
(C) Transcription and translation
(D) Adhesion and invasion

2007
48. Which statement accurately describes an initiating chemical carcinogen?
(A) It stimulates rapid, uncontrolled cell division without damaging DNA
(B) It causes highly reversible epigenetic modifications in target cells
(C) It acts only if applied sequentially after a promoting chemical agent
(D) It induces permanent, irreversible DNA mutations

2011
49. Direct-acting chemical carcinogens differ from indirect-acting (procarcinogens) in that they:
(A) Are electrophiles that do not require metabolic conversion
(B) Require extensive metabolic activation by cytochrome P450 pathways
(C) Only initiate oncogenic processes in the presence of a viral co-factor
(D) Function strictly and exclusively as non-mutagenic tumor promoters

2010
50. Analyze the assertion and reason statements below to select the correct option.
Assertion (A): Tumor promoters induce cancer by causing extensive DNA strand breaks.
Reason (R): Tumor promoters stimulate the clonal expansion of initiated cells without causing new mutations.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2008
51. Aflatoxin B1 is a potent naturally occurring indirect carcinogen primarily associated with the development of:
(A) Hepatocellular carcinoma
(B) Squamous cell carcinoma of the skin
(C) Pulmonary adenocarcinoma
(D) Osteosarcoma

2012
52. Which class of chemical carcinogens is predominantly found in tobacco smoke and smoked meats?
(A) Carcinogenic nitrosamines and nitrosamides
(B) Industrial azo dyes and aromatic amines
(C) Clinically used direct alkylating agents
(D) Polycyclic aromatic hydrocarbons

2001
53. The Ames test is a widely used laboratory assay designed to determine whether a chemical:
(A) Promotes cell division
(B) Causes mutations in bacteria
(C) Induces apoptosis in mammalian cells
(D) Reverses metaplasia

NODIA

2022
54. Prolonged administration of certain therapeutic alkylating agents (e.g., cyclophosphamide) carries the risk of inducing which type of secondary neoplasm?
(A) Deeply situated benign lipomas
(B) High-grade cerebral astrocytomas
(C) Leukemia or lymphoma
(D) Primary skeletal chondrosarcomas

2010
55. Consider the following statements regarding chemical carcinogenesis:
I. Promoters must be applied before initiators to cause tumors.
II. Initiators cause irreversible DNA damage.
III. Promoters cause reversible cellular proliferation.
IV. A single large dose of an initiator guarantees tumor formation without a promoter.
V. Metabolism of procarcinogens often occurs in the liver.
Which of the above statements is/are correct?
(A) II, III and V
(B) I, II, III and V
(C) II, IV and V
(D) I, III and IV

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

2019

37. What does the term "differentiation" mean in the context of neoplastic cells?
(A) The overall speed and frequency at which the tumor cell populations replicate
(B) The extent to which neoplastic cells resemble normal parenchymal cells
(C) The physical ability of the neoplastic cells to synthesize a fibrous capsule
(D) The complex multistep process of migrating and colonizing distant organs

2017

38. A neoplasm described as "anaplastic" exhibits which growth characteristic?
(A) High degree of differentiation
(B) Organized glandular formations
(C) Complete lack of differentiation
(D) Slow, strictly encapsulated expansion

2002

39. Which of the following is typically a characteristic of benign tumors?
(A) Abundant presence of frequent and atypical mitotic figures
(B) Rapid and extensive destruction of surrounding normal tissue
(C) Non-encapsulated, highly infiltrative, and vascularized borders
(D) Expansive growth, compressing surrounding tissue

2003

40. Determine the correct order of progressive cellular changes from normal epithelium to invasive malignancy.
(A) Hyperplasia → Dysplasia → Carcinoma in situ → Invasive carcinoma
(B) Dysplasia → Hyperplasia → Invasive carcinoma → Carcinoma in situ
(C) Carcinoma in situ → Dysplasia → Hyperplasia → Invasive carcinoma
(D) Hyperplasia → Carcinoma in situ → Dysplasia → Invasive carcinoma

2023

41. Cellular pleomorphism, a hallmark of malignancy, refers specifically to:
(A) The abnormal presence of multiple enlarged nucleoli within the cell
(B) Variation in cellular and nuclear size and shape
(C) The rapid, uncontrolled increase in total neoplastic cell numbers
(D) The exclusive formation of massive giant multinucleated tumor cells

2014

42. In a normal cell, the nuclear-to-cytoplasmic (N:C) ratio is typically 1:4 to 1:6. In malignant cells, this ratio commonly shifts towards:
(A) 1:50
(B) 1:20
(C) 1:10
(D) 1:1

2002

43. Nuclear hyperchromasia in malignant cells is primarily attributed to:
(A) Increased DNA content and clumped chromatin
(B) Selective depletion of structural ribosomal RNA
(C) Excessive accumulation of neutral lipids in the nucleus
(D) Infiltration of the nuclear matrix by hemoglobin molecules

2009

44. Atypical mitotic figures, such as tripolar or quadripolar spindles, reliably indicate which phenomenon?
(A) Physiological tissue regeneration
(B) Controlled hyperplastic cell growth
(C) Malignant genomic instability
(D) Reversible metaplastic adaptation

2007

45. Match the cellular hallmarks of malignancy in List I with their accurate descriptions in List II.
List I: (Cellular Hallmarks of Malignancy)
P. Anaplasia
Q. Pleomorphism
R. Hyperchromasia
S. Atypical mitoses
List II: (Descriptions)
1. Complete lack of structural and functional differentiation
2. Significant variation in cellular and nuclear size and shape
3. Intensely dark-staining nuclei due to increased DNA
4. Asymmetrical, multi-polar chromosomal segregation
(A) P-1, Q-2, R-3, S-4
(B) P-2, Q-1, R-4, S-3
(C) P-3, Q-4, R-1, S-2
(D) P-4, Q-3, R-2, S-1

2018

46. What constitutes a functional derangement characteristic of anaplastic tumor cells?
(A) Overproduction of the exact hormone

Click Here to Buy book on Amazon

2021
106. Extravasation of tumor emboli at a distant site mimics which normal physiological process?
(A) Leukocyte adhesion and migration
(B) Erythrocyte lineage maturation
(C) Platelet release and degranulation
(D) Complement pathway activation

2018
107. The "seed and soil" hypothesis of metastasis suggests that:
(A) Disseminated tumor cells colonize randomly, exhibiting equal seeding success and survival in all physiological tissues
(B) Metastasis success depends on tumor cell properties interacting with favorable target organ microenvironments
(C) Tumors grow analogously to plant systems, requiring only basic mechanical support, oxygen, and fluid circulation
(D) Primary tumors release systemic toxic materials that systematically destroy the barriers of all distant organs

2019
108. Dormancy in metastatic tumor cells refers to a state where:
(A) Cells experience immediate and widespread programmed cell death upon reaching a distant secondary site
(B) Cells survive in a non-proliferative state as solitary cells or micrometastases
(C) Cells divide rapidly and uncontrollably while maintaining benign and non-invasive histological features
(D) Cells undergo genetic reversal of all oncogenic mutations to re-establish normal cellular phenotypes

2010
109. Tumor-Specific Antigens (TSA) are characterized by being:
(A) Expressed only by tumor cells and not by any normal host cells
(B) Expressed continuously on both malignant and normal host tissues
(C) Composed of overexpressed versions of normal cellular proteins
(D) Found exclusively on mature, circulating red blood cells

2021
110. Consider the following statements regarding Tumor-Associated Antigens (TAA):
I. They are entirely foreign proteins never seen in the host.
II. They may be normal embryonic proteins expressed inappropriately in adult tumor cells.
III. They can be normal proteins expressed at abnormally high levels.
IV. Alpha-fetoprotein (AFP) is a classic example of a TAA.
Which of the following options identifies the INCORRECT statement(s)?
(A) II and III
(B) I and IV
(C) I only
(D) III and IV

NODIA

2014
111. Carcinoembryonic antigen (CEA) is a well-known marker primarily associated with adenocarcinomas of the:
(A) Brain
(B) Skin
(C) Muscle
(D) Colon

2017
112. Antigens derived from oncogenic viruses (e.g., HPV E6 and E7 proteins) presented on the surface of tumor cells are classified as:
(A) Differentiation antigens
(B) Overexpressed normal antigens
(C) Tumor-specific antigens (TSAs)
(D) Intracellular structural lipids

2021
113. The primary immune effector cells responsible for recognizing and directly killing tumor cells that express foreign peptide antigens on MHC Class I molecules are:
(A) Antigen-presenting B lymphocytes
(B) $CD8^+$ Cytotoxic T lymphocytes (CTLs)
(C) Cytokine-secreting $CD4^+$ T helper cells
(D) Granular polymorphonuclear eosinophils

2007
114. Natural Killer (NK) cells play a crucial role in immune surveillance by specifically targeting tumor cells that have:
(A) Upregulated or high levels of surface MHC Class II expression
(B) Synthesized thick, protective collagenous fibrous capsules
(C) Stopped actively replicating and entered a senescent state
(D) Downregulated or lost surface MHC Class I expression

2016

56. Ultraviolet (UV) radiation primarily damages DNA through the formation of:
(A) Pyrimidine dimers
(B) Double-strand DNA breaks
(C) DNA-protein crosslinks
(D) Single purine deletions

2009

57. Which type of UV radiation is considered the most potent inducer of skin cancers such as squamous cell carcinoma and melanoma?
(A) UVA
(B) UVB
(C) UVC
(D) Vacuum UV

2025

58. Ionizing radiation (e.g., X-rays, gamma rays) promotes carcinogenesis primarily via:
(A) Direct formation of cyclobutane pyrimidine dimers
(B) Generation of ROS and DNA double-strand breaks
(C) Direct binding to and activation of cellular growth receptors
(D) Selective inactivation of the ubiquitin-proteasome system

2019

59. Squamous cell carcinoma in the eyes of Hereford cattle (bovine ocular squamous cell carcinoma) is highly correlated with exposure to:
(A) Prolonged ultraviolet radiation
(B) Chemical fertilizers
(C) Inhaled polycyclic hydrocarbons
(D) Ingested bracken fern

2002

60. Select the correct sequence of events following UV radiation exposure that leads to skin cancer.
(A) UV exposure → Failure of nucleotide excision repair → Thymine dimer formation → Mutation in oncogene → Carcinoma
(B) UV exposure → Thymine dimer formation → Failure of nucleotide excision repair → Mutation in oncogene → Carcinoma
(C) UV exposure → Mutation in oncogene → Thymine dimer formation → Failure of nucleotide excision repair → Carcinoma
(D) Failure of nucleotide excision repair → UV exposure → Thymine dimer formation → Carcinoma → Mutation in oncogene

2006

61. Exposure to radioactive iodine isotopes predominantly increases the risk of which specific neoplasm?
(A) Gastric carcinoma
(B) Renal cell carcinoma
(C) Prostatic adenocarcinoma
(D) Thyroid carcinoma

2003

62. Which human genetic disorder provides a classic model for the effects of defective UV-induced DNA damage repair?
(A) Severe cystic fibrosis
(B) Classical Marfan syndrome
(C) Xeroderma pigmentosum
(D) Huntington's chorea disease

2002

63. Chronic exposure to radon gas is a well-documented risk factor for:
(A) Melanoma
(B) Lung cancer
(C) Osteosarcoma
(D) Leukemia

2006

64. The primary danger of ionizing radiation at the cellular level is its ability to induce:
(A) Aneuploidy through spindle poisoning
(B) Rapid telomere lengthening
(C) Hypermethylation of all cellular oncogenes
(D) Chromosomal translocations and deletions

2008

65. Match the oncogenic viruses in List I with their characteristic associated tumors in List II.
List I: (Oncogenic Viruses)
P. Papillomavirus
Q. Gallid herpesvirus 2
R. Bovine Leukemia Virus (BLV)
S. Feline Leukemia Virus (FeLV)
List II: (Associated Tumors)
1. Marek's disease (T-cell lymphoma in poultry)
2. Enzootic bovine leukosis
3. Cutaneous warts and squamous cell carcinomas
4. Mediastinal lymphoma in cats
(A) P-3, Q-1, R-2, S-4
(B) P-2, Q-3, R-1, S-4
(C) P-1, Q-4, R-3, S-2
(D) P-3, Q-2, R-4, S-1

Downloaded from www.pdf.tube

(C) They are tortuous, leaky, and structurally incomplete
(D) They entirely lack functional lining endothelial cells

2005
87. Cancer cells achieve replicative immortality primarily by avoiding the shortening of:
(A) Centromeres
(B) Spindle fibers
(C) Promoters
(D) Telomeres

2013
88. The Bcl-2 protein contributes to oncogenesis by:
(A) Inhibiting intrinsic apoptosis
(B) Promoting rapid cellular DNA synthesis
(C) Stimulating metastasis to regional lymph nodes
(D) Directly destroying host cytotoxic T cells

2004
89. Hypoxia within the center of a rapidly growing solid tumor directly triggers the expression of which transcription factor to promote angiogenesis?
(A) NF-kB transcription factor
(B) HIF-1a transcription factor
(C) AP-1 transcription factor
(D) STAT3 transcription factor

2011
90. Analyze the assertion and reason statements below to select the correct option.
Assertion (A): Tumor-associated macrophages (TAMs) always function to destroy tumor cells.
Reason (R): TAMs can be recruited from circulating blood monocytes by chemokines secreted by tumor cells.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2016
91. The Warburg effect describes the tendency of cancer cells to rely heavily on which metabolic pathway, even in the presence of adequate oxygen?
(A) Aerobic glycolysis
(B) Oxidative phosphorylation
(C) Beta-oxidation of fatty acids
(D) Gluconeogenesis

2016
92. Carcinomas, which are malignant tumors of epithelial origin, most characteristically disseminate initially via:
(A) Arterial circulation
(B) Cerebrospinal fluid
(C) Lymphatic vessels
(D) Direct seeding of body cavities

2005
93. Sarcomas, arising from mesenchymal tissues, typically favor which initial route of metastasis?
(A) Lymphatic spread
(B) Transcoelomic seeding
(C) Canalicular spread
(D) Hematogenous spread

2013
94. The sentinel lymph node is defined as:
(A) The largest, most anatomically prominent lymph node in the body
(B) The specialized lymph node where native T cells are matured
(C) The first node receiving lymph flow from the primary tumor
(D) A regional lymph node completely replaced by migrating tumor cells

2002
95. Consider the following statements regarding transcoelomic seeding:
I. It occurs when a tumor penetrates the surface of an organ into a natural body cavity.
II. It is a common route for ovarian carcinomas spreading within the peritoneal cavity.
III. It always involves invasion of large arteries.
IV. It frequently leads to neoplastic effusions.
Which of the above statements is/are correct?
(A) I, II and IV
(B) II, III and IV
(C) I and III
(D) I, II and III

NODIA

2011
96. Hematogenous spread of tumors originating in the gastrointestinal tract most frequently results in secondary metastatic deposits in which organ?
(A) Lungs
(B) Brain
(C) Bones
(D) Liver

2013
97. In hematogenous metastasis, tumor emboli in systemic venous circulation (from limbs or head)

(A) Transmembrane growth factor receptor
(B) Tyrosine kinase
(C) Nuclear transcription factor
(D) Cytoplasmic second messenger

2019
77. Which of the following defines a tumor suppressor gene?
(A) A gene that actively protects tumor cells from the host immune system
(B) A viral gene that successfully suppresses host apoptotic cell death
(C) A gene that promotes the rapid formation of new tumor blood vessels
(D) A gene that inhibits cell growth or promotes apoptosis

2018
78. The Retinoblastoma (Rb) protein regulates the cell cycle by controlling transition at which key checkpoint?
(A) S to G2 phase
(B) G2 to M phase
(C) G1 to S phase
(D) M to G1 phase

2012
79. The p53 gene is frequently termed the "guardian of the genome" because its normal function is to:
(A) Actively enhance total DNA replication speed and accuracy
(B) Arrest the cell cycle for DNA repair or trigger apoptosis
(C) Prevent the progressive enzymatic shortening of telomeres
(D) Actively promote angiogenesis around local DNA repair sites

2010
80. Determine the correct order of events mediated by normal p53 following DNA damage.
(A) p53 accumulation → DNA damage → p21 transcription → Cell cycle arrest
(B) DNA damage → p53 accumulation → p21 transcription → Cell cycle arrest
(C) p21 transcription → DNA damage → p53 accumulation → Cell cycle arrest
(D) Cell cycle arrest → DNA damage → p53 accumulation → p21 transcription

2014
81. A mutation resulting in the loss of heterozygosity (LOH) is most relevant to the functional inactivation of:
(A) Tumor suppressor genes
(B) Active viral oncogenes
(C) Cellular proto-oncogenes
(D) Activated telomerase genes

2005
82. Which genetic alteration mechanism is most responsible for the activation of the c-myc oncogene in Burkitt lymphoma?
(A) Somatic point mutation
(B) Focal gene amplification
(C) Chromosomal translocation
(D) Nonsense gene mutation

2022
83. Tumor angiogenesis is essential for a neoplasm to grow beyond approximately what size?
(A) 1-2 millimeters
(B) 1-2 centimeters
(C) 5-10 millimeters
(D) 5-10 centimeters

2018
84. The most critical pro-angiogenic factor secreted by tumor cells to stimulate blood vessel growth is:
(A) Tumor Necrosis Factor Alpha
(B) Transforming Growth Factor Beta
(C) Lymphocyte-derived Interleukin-2
(D) Vascular Endothelial Growth Factor

2021
85. Match the tumor biological concepts in List I with their mechanisms in List II.
List I: (Tumor Biological Concepts)
P. Angiogenesis
Q. Cellular immortality
R. Apoptosis evasion
S. Immune evasion
List II: (Mechanisms)
1. Reactivation of telomerase to maintain chromosomal ends
2. Secretion of VEGF to form new blood vessels
3. Overexpression of Bcl-2 to prevent cell death
4. Downregulation of MHC class I molecules
(A) P-2, Q-1, R-3, S-4
(B) P-1, Q-2, R-4, S-3
(C) P-3, Q-4, R-1, S-2
(D) P-2, Q-3, R-1, S-4

2010
86. How do newly formed tumor blood vessels structurally differ from normal physiological blood vessels?
(A) They possess significantly thicker muscular arterial walls
(B) They are highly organized, symmetrical, and hierarchical

Click Here to Buy book on Flipkart

2014

66. Oncogenic DNA viruses typically promote tumorigenesis by expressing viral proteins that bind and inactivate which host cellular proteins?
(A) Host cytochrome P450 metabolizing enzymes
(B) Host nuclear DNA replication polymerases
(C) Tumor suppressor proteins like p53 and Rb
(D) Intra-lysosomal acidic hydrolase enzymes

2012

67. The E6 protein of high-risk Papillomaviruses contributes to oncogenesis by directly mediating the degradation of:
(A) p53 protein
(B) Retinoblastoma (Rb) protein
(C) Ras protein
(D) Myc protein

2016

68. Gallid herpesvirus 2 (Marek's disease virus) induces neoplastic transformation primarily in which cell type?
(A) B lymphocytes
(B) Macrophages
(C) Erythrocytes
(D) T lymphocytes

2010

69. Retroviruses like Feline Leukemia Virus (FeLV) predominantly cause cancer through which mechanism?
(A) Lysis of target cells leading to compensatory hyperplasia
(B) Insertional mutagenesis near a host proto-oncogene
(C) Expression of large DNA-binding antigens
(D) Induction of massive chromosomal translocations

2013

70. Analyze the assertion and reason statements below to select the correct option.
Assertion (A): Most RNA tumor viruses integrate their genome directly into host RNA to cause cancer.
Reason (R): Retroviruses use reverse transcriptase to synthesize a proviral DNA copy that integrates into the host cell genome.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2002

71. Bovine Leukemia Virus (BLV) causes Enzootic Bovine Leukosis, which is characterized by the neoplastic proliferation of:
(A) T lymphocytes
(B) Neutrophils
(C) Eosinophils
(D) B lymphocytes

2019

72. Which viral gene is primarily responsible for the transactivating function in the Bovine Leukemia Virus, leading to cellular proliferation?
(A) tax
(B) gag
(C) pol
(D) env

2012

73. Feline Immunodeficiency Virus (FIV) increases the risk of lymphoma primarily via:
(A) Carrying a highly active transforming viral oncogene
(B) Inducing severe systemic immunosuppression
(C) Direct insertional activation of the host myc gene
(D) Stimulating overproduction of feline parathyroid hormone

2010

74. Proto-oncogenes are best described as:
(A) Mutated genes that actively suppress cellular growth
(B) Normal cellular genes promoting cell growth
(C) Foreign viral genes integrated into the host genome
(D) Genes directly responsible for initiating apoptosis

NODIA

2005

75. Consider the following statements regarding the ras gene:
I. It is a proto-oncogene.
II. Its mutated form continuously binds GDP and remains inactive.
III. It encodes a small G-protein involved in signal transduction.
Which of the above statements is/are incorrect?
(A) I and III
(B) II only
(C) I and II
(D) III only

2018

76. The myc oncogene promotes tumorigenesis primarily functioning as a:

most typically lodge first in the capillary beds of the:
(A) Lungs
(B) Kidneys
(C) Spleen
(D) Liver

2012
98. Why is the spleen rarely a site of gross secondary metastasis despite receiving massive blood flow?
(A) The splenic parenchyma completely lacks a normal capillary network
(B) Splenic tissue actively secretes a specific anti-neoplastic hormone
(C) Abundant splenic immune cells and harsh hemodynamics
(D) The main splenic artery is structurally unable to transport tumor emboli

2019
99. "Skip metastasis" in the lymphatic system refers to a situation where:
(A) A tumor bypasses regional lymph nodes to form metastases in more distant lymph nodes
(B) Tumor cells completely bypass regional lymph nodes and directly enter veins
(C) Tumor cells physically destroy the lymphatic vessels causing localized edema
(D) Tumor cells completely revert back to a benign phenotypic status in the node

2009
100. Consider the following statements about lymphatic dissemination:
I. It is the favored route for fibrosarcomas.
II. The subcapsular sinus of the lymph node is often the first site of metastatic growth.
III. Enlarged lymph nodes near a tumor always indicate metastasis.
IV. Tumor cells enter lymphatic capillaries easier than arteries due to lack of a basement membrane.
V. Lymphatic vessels can become physically obstructed by tumor emboli.
Which of the above statements is/are incorrect?
(A) I and III
(B) II, IV and V
(C) I, III and V
(D) II and IV

2013
101. The metastatic cascade involves several sequential steps. Which of the following is an essential requirement for a carcinoma to initiate local invasion?
(A) Development of a thick fibrous capsule
(B) Loss of E-cadherin mediated cell-cell adhesion
(C) Maturation of tumor cells into squamous epithelium
(D) Inactivation of all cellular proteases

2009
102. Degradation of the extracellular matrix (ECM) and basement membrane by invading tumor cells is primarily facilitated by:
(A) De novo synthesis of dense structural collagen fibers
(B) Intracellular enhanced production of functional E-cadherins
(C) Secretion of Matrix Metalloproteinases (MMPs)
(D) Overexpression of wild-type p53 tumor suppressor protein

2025
103. Intravasation is the process by which tumor cells:
(A) Exit a blood vessel lumen to establish a distant secondary tumor
(B) Form a completely new blood vessel from pre-existing host capillaries
(C) Strongly adhere to structural components of the extracellular matrix
(D) Penetrate a vessel wall to enter circulation

2006
104. Once inside the bloodstream, tumor cells often form emboli by aggregating with which host cells to protect against shear stress and immune destruction?
(A) Platelets and coagulation factors
(B) Circulating mature erythrocytes
(C) Granulocytic host eosinophils
(D) Differentiated brain astrocytes

2024
105. Consider the following statements regarding the metastatic cascade:
I. Intravasation precedes extravasation.
II. Tumor cells must survive the physical shear stress of the circulation.
III. All cells that enter the circulation successfully form secondary tumors.
Which of the above statements is/are correct?
(A) I and II
(B) I and III
(C) II and III
(D) I only

NODIA

(D) Proliferation of hepatocytes to replace necrotic liver tissue

2025

27. Why is dysplasia generally considered a precancerous lesion?
(A) It has genetic and morphologic alterations that can progress to carcinoma
(B) It invariably invades through the underlying basement membrane into deeper tissues
(C) It causes rapid and immediate systemic metastasis to distant regional lymph nodes
(D) It actively suppresses the host's cellular and humoral immune system responses

2011

28. Which of the following is the standard suffix used to denote a benign tumor of mesenchymal origin?
(A) -carcinoma
(B) -sarcoma
(C) -oma
(D) -plasia

2024

29. A malignant neoplasm arising from glandular epithelium is correctly termed a:
(A) Squamous cell carcinoma
(B) Adenoma
(C) Fibrosarcoma
(D) Adenocarcinoma

2002

30. Analyze the assertion and reason statements below to select the correct option.
Assertion (A): Lymphoma is a malignant tumor of lymphocytes.
Reason (R): The suffix "-oma" always indicates a benign neoplasm without exception.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2003

31. Which of the following tumor names represents an exception to standard nomenclature by denoting a highly malignant tumor despite ending in "-oma"?
(A) Fibroma
(B) Melanoma
(C) Lipoma
(D) Osteoma

2007

32. A benign neoplasm composed of smooth muscle is termed a:
(A) Rhabdomyoma
(B) Leiomyosarcoma
(C) Rhabdomyosarcoma
(D) Leiomyoma

2017

33. A teratoma is distinguished from other neoplasms by which unique characteristic?
(A) It contains cells from more than one germ layer, often all three
(B) It arises exclusively from cells of a single embryological germ layer
(C) It is invariably malignant and exhibits rapid metastatic dissemination
(D) It occurs exclusively within the tissues of the central nervous system

2017

34. What term is used to describe a mass of disorganized but mature indigenous tissue present at a normal anatomical site?
(A) Choristoma
(B) Teratoma
(C) Hamartoma
(D) Sarcoma

2019

35. Consider the following statements regarding malignant mesenchymal tumors:
I. They are broadly referred to as sarcomas.
II. A malignant tumor of cartilage is called an osteosarcoma.
III. A malignant tumor of adipose tissue is called a liposarcoma.
IV. They typically spread via lymphatic vessels in their early stages.
Which of the above statements is/are incorrect?
(A) I and III
(B) II and IV
(C) I, II and IV
(D) I, III and IV

2024

36. Which feature strongly differentiates a malignant neoplasm from a benign one?
(A) Presence of a highly protective, well-defined fibrous capsule
(B) Relatively slow, strictly expansive rate of cellular growth
(C) Close morphological resemblance to the normal tissue of origin
(D) Ability to invade local tissues and metastasize

Downloaded from www.pdf.tube

2005

115. Consider the following mechanisms by which tumors evade immune destruction:

I. Downregulation of MHC Class I molecules.
II. Secretion of immunosuppressive cytokines like TGF-beta.
III. Upregulation of strong tumor-specific antigens.
IV. Expression of immune checkpoint molecules like PD-L1.
V. Induction of regulatory T cells (Tregs).

Which of the following is the correct combination of statements?

(A) I, II, IV and V
(B) I, II, III and V
(C) I, III, IV and V
(D) II, III, IV and V

2013

116. How does the expression of Programmed Death-Ligand 1 (PD-L1) on a tumor cell surface protect it from the immune system?

(A) It acts as a physical barrier that mechanically blocks host antibodies and complement proteins from binding
(B) It triggers intracellular signaling cascades causing the tumor cell to undergo immediate apoptotic death
(C) It binds PD-1 on T cells, sending inhibitory signals that halt T cell attack
(D) It releases specialized proteolytic enzymes that rapidly degrade the surrounding extracellular matrix

2025

117. Macrophages activated by the tumor microenvironment (M2 phenotype TAMs) generally contribute to tumor progression by:

(A) Secreting growth factors, promoting angiogenesis, and suppressing immunity
(B) Releasing massive quantities of cytotoxic nitric oxide to kill adjacent cells
(C) Phagocytosing viable and metabolically active tumor cells at a high rate
(D) Differentiating directly into antigen-specific $CD8^+$ cytotoxic T cells

2019

118. Which structural barrier can physically protect solid tumors from immune cell infiltration?

(A) Viscous surfactant layer
(B) Neuronal myelin sheath
(C) Fibrous periosteal membrane
(D) Dense desmoplastic stroma

2011

119. Antibody-Dependent Cellular Cytotoxicity (ADCC) against tumors is primarily mediated by which cell type binding to the Fc region of tumor-bound antibodies?

(A) Polymorphonuclear neutrophils
(B) Cytotoxic Natural Killer (NK) cells
(C) Circulating granular basophils
(D) Tissue-resident mast cells

2019

120. Cancer cachexia, characterized by progressive loss of body fat and lean body mass, is primarily driven by:

(A) Chronically reduced nutritional intake and poor dietary absorption alone
(B) Massive, acute bleeding into the upper gastrointestinal tract pathways
(C) Systemic release of cytokines like TNF-alpha by the tumor and host
(D) Progressive mechanical obstruction of the proximal esophagus passage

NODIA

2023

121. Anemia of chronic disease in cancer patients is largely mediated by elevated levels of hepcidin, which functions by:

(A) Stimulating splenic macrophages to destroy muscle cells
(B) Preventing renal cortical cells from producing erythropoietin
(C) Inhibiting pluripotential bone marrow stem cells directly
(D) Inhibiting iron absorption and locking iron within macrophages

2001

122. Paraneoplastic syndromes are defined as systemic symptom complexes in cancer patients that:

(A) Are independent of local tumor growth, metastasis, or indigenous hormone production
(B) Result directly from the physical mass effect and mechanical pressure of the primary tumor
(C) Develop when distant metastatic growths compress or infiltrate adjacent peripheral nerves
(D) Represent toxic physiological side effects occurring solely as an adverse reaction to chemotherapy

2005

123. A classic example of an endocrinopathy paraneoplastic syndrome is hypercalcemia of

malignancy, often caused by the tumor's ectopic secretion of:
(A) Epithelial-derived pancreatic insulin hormone
(B) Parathyroid Hormone-Related Protein (PTHrP)
(C) Thyroid C-cell-derived calcitonin hormone
(D) Pancreatic alpha-cell-derived glucagon hormone

2013
124. Hypertrophic osteopathy, a paraneoplastic syndrome characterized by periosteal new bone formation in the distal limbs, is most frequently associated with neoplasms of the:
(A) Liver
(B) Lungs or thoracic cavity
(C) Central Nervous System
(D) Small intestine

2024
125. Trousseau's syndrome is a paraneoplastic condition involving which bodily system?
(A) Coagulation system (migratory thrombophlebitis)
(B) Nervous system (progressive peripheral neuropathy)
(C) Endocrine system (refractoglycemia)
(D) Integumentary system (acanthosis nigricans)

2007
126. A dog presents with a functional mast cell tumor. The massive release of histamine from this tumor can lead to which significant systemic complication?
(A) Severe hypertension
(B) Hypoglycemia
(C) Gastric ulceration
(D) Hypercalcemia

2004
127. Tumor grading is a histopathological assessment primarily based on:
(A) The total physical size and volume of the primary tumor mass
(B) The total number and location of clinically involved lymph nodes
(C) The confirmed presence or absence of distant organ metastasis
(D) Cellular differentiation and mitotic activity of the tumor cells

2001
128. In the TNM clinical staging system, the "T" evaluates:
(A) Histological tumor grade and cellular differentiation
(B) Chronological time elapsed since clinical onset of symptoms
(C) Physical size and local invasiveness of the primary tumor
(D) Total cumulative number of distinct tumors in the host body

2017
129. What does the designation "N0" imply in the TNM staging system?
(A) Complete absence of the primary tumor
(B) No regional lymph node metastasis
(C) Extensive regional lymph node involvement
(D) Necrosis present within the tumor

2003
130. A tumor staged as T3, N2, M1 indicates:
(A) A small localized tumor with no nodal involvement or metastasis
(B) A pre-invasive carcinoma in situ confined to epithelial layers
(C) A benign neoplasm with localized expansion and no invasion
(D) A large tumor with regional nodal involvement and metastasis

2017
131. Which of the following statements best contrasts tumor grading and staging?
(A) Grading evaluates cellular behavior; staging measures anatomical disease extent
(B) Grading is primarily a clinical determination; staging is purely microscopic
(C) Staging determines specific histological cell types; grading measures tumor size
(D) Grading is utilized only for benign tumors; staging is for malignant tumors only

2016
132. Exfoliative cytology relies on the examination of cells that have:
(A) Been surgically excised as a large solid block of tissue
(B) Been mechanically extracted using a hollow core biopsy needle
(C) Spontaneously shed from epithelial surfaces or into body fluids
(D) Been chemically fixed and frozen immediately post-mortem

2008
133. Fine Needle Aspiration Cytology (FNAC) is highly useful for rapidly evaluating:
(A) Individual cell morphology of a palpable mass via a thin needle

Click Here to Buy book on Amazon

(B) The deep histological architectural framework of a solid organ
(C) Fully mapped excisional surgical margins of a solid tumor mass
(D) The precise depth of tumor invasion into the basement membrane

2022
134. Which diagnostic procedure is considered the "gold standard" for definitively determining both tumor type and margins of excision?
(A) Fine needle aspiration cytology of solid tissue masses
(B) Exfoliative cytological analysis of pleural effusions
(C) Serum-based biochemical tumor marker screening assays
(D) Surgical histopathology of an excisional tissue biopsy

2007
135. The primary disadvantage of cytology (FNAC) compared to histopathology is:
(A) Cytology routinely requires deep general patient anesthesia
(B) Cytology lacks evaluation of tissue architecture and invasion
(C) Cytology requires specialized laboratories and weeks to process
(D) Cytology is significantly more expensive than biopsy methods

2009
136. Routine tissue processing for histopathology requires fixation, most standardly performed using:
(A) Dehydrated 100% ethanol solution
(B) Buffered glutaraldehyde fixative
(C) 10% Neutral Buffered Formalin
(D) Isotonic normal saline solution

2007
137. The standard, universal stain used for evaluating routine surgical histopathology sections is:
(A) Periodic acid-Schiff (PAS)
(B) Toluidine Blue
(C) Ziehl-Neelsen
(D) Hematoxylin and Eosin (H&E)

2011
138. Immunohistochemistry (IHC) identifies specific cell lineage markers by utilizing:
(A) Chromogen-tagged antibodies binding to specific cellular antigens
(B) Heavy metal-based histological dyes that bind directly to nucleic acids
(C) Synthesized fluorescent probes that hybridize to cellular target RNA
(D) Endogenous heat-induced autofluorescence signals of cellular proteins

2021
139. In a poorly differentiated tumor, a positive immunohistochemical stain for Cytokeratin definitively identifies the tumor as being of which origin?
(A) Mesenchymal (Sarcoma)
(B) Epithelial (Carcinoma)
(C) Leukocytic (Lymphoma)
(D) Melanocytic (Melanoma)

2005
140. Vimentin is an intermediate filament protein commonly used as an immunohistochemical marker for tumors of which origin?
(A) Epithelial
(B) Mesenchymal
(C) Neuroendocrine
(D) Glial

2010
141. The diagnostic application of the Polymerase Chain Reaction (PCR) in oncology is particularly useful for identifying:
(A) Specific genetic mutations, translocations, or viral DNA
(B) The exact physical dimensions and total volume of the tumor
(C) The macroscopic color and consistency of the stromal tissue
(D) The exact degree of clinical edema in surrounding tissues

NODIA

2017
142. Which molecular technique is best suited to visualize specific gene amplifications (e.g., HER2) directly within intact tissue sections?
(A) Flow cytometry
(B) Western blotting
(C) Fluorescence In Situ Hybridization (FISH)
(D) Enzyme-Linked Immunosorbent Assay (ELISA)

2014
143. Flow cytometry is an advanced diagnostic tool most routinely applied for the subtyping and diagnosis of:
(A) Malignant primary osteosarcomas of the bone
(B) Infiltrative squamous cell carcinomas of the skin
(C) Benign or atypical meningiomas of the brain

2001

208. The most significant prognostic indicator for a patient with a localized malignant melanoma is typically the:
(A) The horizontal width of the primary tumor
(B) The pigmentation and color of the tumor mass
(C) The histological depth of tumor invasion
(D) The physical presence of local inflammation

2016

209. Cytological evaluation of a lipoma via fine needle aspiration will typically reveal:
(A) Dense, highly cohesive clusters of anaplastic epithelial cells
(B) Free lipid droplets and uniform fat-filled cells with small nuclei
(C) Abundant cytological metachromatic purple cytoplasmic granules
(D) Anaplastic spindle cells producing dense intercellular collagen

2022

210. Which diagnostic marker is most appropriate to confirm the epithelial origin of an anaplastic tumor using immunohistochemistry?
(A) Vimentin
(B) CD3
(C) Desmin
(D) Cytokeratin

****

Downloaded from www.pdf.tube

2024

155. Lipomas are commonly encountered benign tumors composed of mature:
(A) Fibroblasts
(B) Adipocytes
(C) Chondrocytes
(D) Histiocytes

2004

156. A lymphoma is considered a specialized tumor because it involves the malignant proliferation of lymphocytes occurring primarily in:
(A) Circulating blood and primary bone marrow compartments
(B) Lymph nodes, spleen, and mucosa-associated lymphoid tissue
(C) Isolated brain and spinal cord meningeal structures
(D) Epithelial layers and adjacent cutaneous epidermis

2015

157. Mast cell tumors are particularly notorious in veterinary pathology for their variable behavior and the cytoplasmic presence of granules containing:
(A) Histamine, heparin, and proteolytic enzymes
(B) Neutral glycogen and acidic epithelial mucin
(C) Dark melanin and brown hemosiderin pigments
(D) Insoluble calcium and structural phosphorus

2023

158. The standard stain used to best highlight the metachromatic granules of a mast cell tumor is:
(A) Acid-fast Congo Red stain
(B) Masson's Trichrome stain
(C) Toluidine Blue or Giemsa
(D) Lipophilic Oil Red O stain

2017

159. Melanomas arise from melanocytes and can be benign or highly malignant. The highest rate of malignancy for melanomas in dogs is associated with which anatomical location?
(A) Haired skin covering the limbs or trunk
(B) Distal aspects of the external ear pinna
(C) Thick plantar aspects of the canine footpads
(D) Oral cavity and mucocutaneous junctions

2008

160. Canine Transmissible Venereal Tumor (CTVT) is unique among neoplasms because it is:
(A) Caused by a highly contagious and oncogenic retrovirus genome
(B) Restricted solely to geriatric and severely immunocompromised canines
(C) An allograft transmitted by physical transfer of living tumor cells
(D) Highly lethal and always fatal within three weeks of initial onset

NODIA

2001

161. Histiocytomas are common, rapid-growing benign skin tumors in young dogs that frequently exhibit which unusual biological behavior?
(A) Rapid transformation into highly malignant histiocytic sarcomas
(B) Spontaneous regression driven by infiltrating host T cells
(C) Systemic metastasis followed by a rapid, spontaneous clinical cure
(D) Massive intracellular production and accumulation of hard keratin

2007

162. Plasmacytomas are specialized tumors derived from B-cell lineages. A malignant systemic proliferation of these cells (Multiple Myeloma) is often associated with the presence of which marker in urine?
(A) Conjugated urobilinogen
(B) Acetoacetate ketones
(C) Filtered excess glucose
(D) Bence Jones proteins

2025

163. What is the primary distinguishing microscopic feature of an adenoma compared to an adenocarcinoma?
(A) Adenomas exhibit organized architecture lacking stromal/vascular invasion
(B) Adenomas form clear glands; adenocarcinomas form solid sheets only
(C) Adenomas routinely demonstrate a highly elevated cellular mitotic index
(D) Adenocarcinomas completely lose the cellular ability to produce mucin

2025

164. In determining the prognosis of a mast cell tumor, the Patnaik or Kiupel grading systems primarily evaluate:
(A) The amount of histamine released into the blood
(B) The physical diameter of the gross tumor
(C) Cellular pleomorphism, mitotic count, and invasiveness
(D) The age of the affected animal

2019

165. An amelanotic melanoma is a variant that is challenging to diagnose on standard H&E because:
(A) The cells lack the characteristic melanin pigment
(B) The cells are completely anucleated
(C) The cells rapidly undergo apoptosis during fixation
(D) The tumor forms abundant bone matrix

2024

166. Diagnostic confirmation of an amelanotic melanoma usually requires immunohistochemical staining for:
(A) Cytokeratin
(B) CD3
(C) Vimentin only
(D) Melan-A or PNL2

2017

167. Which of the following factors plays a critical role in the spontaneous regression of Canine Transmissible Venereal Tumor (CTVT)?
(A) Progressive cellular exhaustion of internal tumor telomerase enzymes
(B) A robust host cell-mediated immune response targeting tumor cells
(C) Localized ischemic necrosis secondary to failed tumor angiogenesis
(D) High titers of virus-neutralizing antibodies produced by the host

2009

168. The characteristic chromosomal abnormality observed in the cells of Canine Transmissible Venereal Tumor (CTVT) consists of:
(A) Exactly 78 standard, structurally intact canine chromosomes
(B) Massive polyploidy exceeding 150 individual chromosomes
(C) Typically 57 to 64 abnormal and rearranged chromosomes
(D) A single, highly stable ring-shaped chromosome structure

2018

169. Soft tissue sarcomas are a group of mesenchymal tumors grouped together clinically because they share similar biological behaviors, including:
(A) Early and widespread metastasis through host lymphatic vessels
(B) Neoplastic developments occurring exclusively within bone tissues
(C) High clinical responsiveness to low-dose external radiation therapy
(D) High local invasiveness and recurrence with late or rare metastasis

2009

170. Which of the following represents a characteristic feature of a peripheral nerve sheath tumor (e.g., Schwannoma)?
(A) Antoni A and Antoni B tissue patterns
(B) Abundant keratin pearl formation
(C) High levels of estrogen receptors
(D) Massive production of histamine

2012

171. A tumor arising from the lining cells of the pleural or peritoneal cavity, often linked to asbestos exposure in humans but also occurring in animals, is termed:
(A) Endothelioma
(B) Mesothelioma
(C) Synovial sarcoma
(D) Rhabdomyoma

2013

172. Epulides (e.g., peripheral odontogenic fibromas) are common benign tumors located strictly in the:
(A) Skin of the digits
(B) Gingiva of the oral cavity
(C) Lining of the esophagus
(D) Mucosa of the stomach

2004

173. What is the defining morphological feature of a hemangiopericytoma?
(A) Spindle cells in fingerprint whorls surrounding small vessels
(B) Uniform palisading arrays of neoplastic cells forming true rosettes
(C) Huge multinucleated osteoclast-like giant cells with bone fragments
(D) Discrete nests of round cells separated by dense collagenous bands

2001

174. Cholangiocarcinomas arise from the epithelial cells of the:
(A) Intestinal crypts
(B) Pancreatic acini
(C) Bile ducts
(D) Gastric mucosa

2015

175. Which type of necrosis is classically associated with rapidly growing solid tumors due to an inadequate blood supply?
(A) Liquefactive necrosis

Click Here to Buy book on Flipkart

(B) Caseous necrosis
(C) Fat necrosis
(D) Coagulative necrosis

2008

176. Desmoplasia, the dense fibrous stromal reaction induced by some tumors, is most commonly associated with:
(A) Malignant lymphomas
(B) Benign lipomas
(C) Scirrhous carcinomas
(D) Vascular hemangiomas

2025

177. A definitive diagnosis of amyloidosis associated with multiple myeloma relies on confirming apple-green birefringence using which stain under polarized light?
(A) Periodic acid-Schiff
(B) Congo Red
(C) Von Kossa
(D) Perl's Prussian Blue

2023

178. The mechanism by which ionizing radiation selectively kills tumor cells relies on targeting cells that are:
(A) Locked in a prolonged, non-proliferative G0 resting phase
(B) Highly differentiated and metabolically specialized
(C) Devoid of functional mitochondria and metabolic pathways
(D) Actively dividing with diminished DNA repair capacity

2024

179. A key characteristic that allows malignant tumors to bypass the Hayflick limit of cellular division is the reactivation of:
(A) Telomerase
(B) Cytochrome C
(C) Caspase-9
(D) Helicase

2012

180. Hypercalcemia of malignancy in dogs is most classically associated with which specific tumor type?
(A) Well-differentiated dermal lipoma
(B) Benign cavernous splenic hemangioma
(C) Apocrine gland anal sac adenocarcinoma
(D) Malignant intraoral amelanotic melanoma

2021

181. Myasthenia gravis is a paraneoplastic syndrome most commonly associated with which specific neoplasm?
(A) Thymoma
(B) Hepatoma
(C) Mast cell tumor
(D) Osteosarcoma

2006

182. Which cellular adaptation is characterized by an increase in the size of cells due to the synthesis of more structural components, without cellular division?
(A) Hyperplasia
(B) Metaplasia
(C) Dysplasia
(D) Hypertrophy

2021

183. Hypoplasia differs from atrophy because in hypoplasia, the organ:
(A) Was fully developed and subsequently shrank in size
(B) Never reached normal mature size during development
(C) Underwent localized, aggressive malignant transformation
(D) Is completely congenitally absent from the time of birth

2012

184. The physiological enlargement of the uterus during pregnancy is a classic example of:
(A) Cellular hypertrophy alone
(B) Cellular hyperplasia alone
(C) Both hypertrophy and hyperplasia
(D) Epithelial metaplasia alone

2014

185. Chronic irritation of the respiratory mucosa in a smoker leads to the replacement of ciliated columnar epithelium by stratified squamous epithelium. This is termed:
(A) Squamous dysplasia
(B) Squamous hyperplasia
(C) Squamous carcinoma
(D) Squamous metaplasia

2002

186. Atrophy of a skeletal muscle following a cast application for a bone fracture is classified as:
(A) Disuse atrophy
(B) Denervation atrophy
(C) Endocrine atrophy
(D) Ischemic atrophy

Downloaded from www.pdf.tube

(D) Hematopoietic neoplasms like leukemia and lymphoma

2013

144. Papillomas are benign epithelial neoplasms characteristically exhibiting what type of gross growth pattern?
(A) Deeply infiltrative and ulcerated lesions
(B) Thin-walled, cystic, and fluid-filled masses
(C) Exophytic, branching, and cauliflower-like growths
(D) Flat, highly scirrhous, and firm plaques

2011

145. Squamous cell carcinomas frequently produce microscopic structures formed by concentric layers of keratinizing cells known as:
(A) Call-Exner bodies
(B) Keratin pearls
(C) Homer Wright rosettes
(D) Psammoma bodies

2017

146. A scirrhous adenocarcinoma is characterized morphologically by:
(A) A very soft, brain-like gross tissue consistency
(B) Large, multi-focal areas of internal hemorrhage
(C) A complete histological lack of glandular structures
(D) Abundant, dense fibrous connective tissue stroma

2015

147. Urothelial (transitional cell) carcinomas are most commonly located in the:
(A) Urinary bladder
(B) Renal cortex
(C) Gallbladder
(D) Trachea

2002

148. Basal cell tumors in animals typically present as:
(A) Aggressive and rapidly metastatic lesions of the lung
(B) Diffuse, severe thickening of the intestinal mucosa
(C) Solitary, discrete, and benign nodules in the skin
(D) Highly malignant, cellular effusions in the thorax

2025

149. Sebaceous adenomas are common, benign glandular tumors arising from the:
(A) Adnexal glands of the skin
(B) Major and minor salivary glands
(C) Exocrine lobules of the pancreas
(D) Bilobed canine prostate gland

2008

150. Fibrosarcomas are malignant mesenchymal tumors that are microscopically characterized by:
(A) Large round sheets of cells with prominent cytoplasmic granules
(B) Nests of uniform polyhedral cells separated by thick fibrous septa
(C) Widespread histological formation of disorganized osteoid or bone
(D) Plump spindle cells arranged in interlacing herringbone patterns

2024

151. Osteosarcomas are highly aggressive malignant tumors primarily identified by the neoplastic cells' ability to produce:
(A) Hyaline and fibrous cartilage
(B) Osteoid or unmineralized matrix
(C) Dense, parallel collagen bundles
(D) Dark, granular melanin pigment

2004

152. In veterinary oncology, appendicular osteosarcomas in large breed dogs frequently metastasize early to the:
(A) Liver
(B) Spleen
(C) Lungs
(D) Brain

2006

153. Hemangiosarcomas are malignant tumors arising from:
(A) Cardiac muscle cells
(B) Smooth muscle of blood vessels
(C) Hematopoietic stem cells
(D) Vascular endothelial cells

2021

154. A common and life-threatening presentation of a splenic hemangiosarcoma is:
(A) Acute tumor rupture causing massive hemoabdomen
(B) Gradual obstruction of the gastrointestinal tract
(C) Severe hypercalcemia leading to renal failure
(D) Overproduction of red blood cells (polycythemia)

NODIA

polyclonal population (reactive hyperplasia) by analyzing antigen receptor gene rearrangements?
(A) Western Blotting
(B) PCR for Antigen Receptor Rearrangement (PARR)
(C) Immunohistochemistry (IHC)
(D) Enzyme-Linked Immunosorbent Assay (ELISA)

2019
198. Mast cell tumors are routinely graded to predict biological behavior. Which characteristic is typically evaluated during histopathological grading?
(A) Total amount of dark intracellular melanin pigment
(B) Relative thickness of the protective tumor capsule
(C) Number of vascular blood vessels entering the tumor
(D) Depth of invasion, mitoses, and cell morphology

2018
199. A diagnostic finding of "pleomorphism" on a cytology report implies that the cells show:
(A) High affinity for basic stains
(B) Variability in size and shape
(C) Multiple distinct nuclei
(D) Complete uniformity

2014
200. Which of the following pro-angiogenic factors is primarily secreted by hypoxic tumor cells to stimulate the migration and proliferation of endothelial cells?
(A) Vascular Endothelial Growth Factor (VEGF)
(B) Transforming Growth Factor-beta (TGF-β)
(C) Tumor Necrosis Factor-alpha (TNF-α)
(D) Interferon-gamma (IFN-γ)

2023
201. Which specific molecule do tumor cells typically secrete to break down the basement membrane and facilitate invasion?
(A) Systemic serum albumin
(B) Active myeloperoxidase
(C) Circulating fibrinogen
(D) Metalloproteinases (MMPs)

2006
202. Why is the lung the most common site for hematogenous metastasis from many solid tumors?
(A) The lung provides the first major capillary bed for venous drainage
(B) Lung tissues secrete specific chemokines that actively attract tumor cells
(C) Local oxygen tension in pulmonary tissues forces tumor cells to arrest
(D) Pulmonary alveolar microenvironments completely lack any immune response

2016
203. Carcinomas of the ovary frequently spread extensively along the peritoneal surfaces without necessarily invading deeply into organs. This is an example of:
(A) Hematogenous spread
(B) Transcoelomic seeding
(C) Lymphatic permeation
(D) Canalicular spread

2024
204. "Anemia of chronic disease" associated with neoplasia is characterized by a lab profile showing:
(A) Low serum iron, high iron-binding capacity, and low storage ferritin
(B) Low serum iron, low iron-binding capacity, and high storage ferritin
(C) High serum iron, low iron-binding capacity, and low storage ferritin
(D) High serum iron, high iron-binding capacity, and high storage ferritin

2007
205. Hypoglycemia can occur as a paraneoplastic syndrome most notably with which type of tumor?
(A) Pancreatic beta-cell insulinoma
(B) Cutaneous amelanotic melanoma
(C) Invasive squamous cell carcinoma
(D) Poorly-differentiated mast cell tumor

2025
206. A developmental anomaly characterized by a disorganized mass of mature, specialized tissues indigenous to the particular anatomical site of occurrence is termed a:
(A) Choristoma
(B) Hamartoma
(C) Teratoma
(D) Myxoma

2022
207. A malignant tumor composed of cartilage is known as a:
(A) Chondroma
(B) Osteosarcoma
(C) Fibrosarcoma
(D) Chondrosarcoma

Click Here to Buy book on Amazon

2018

187. Reversible loss of cell structural uniformity and architectural orientation within an epithelium is defined as:
(A) Hyperplasia
(B) Dysplasia
(C) Anaplasia
(D) Metaplasia

2018

188. Which of the following is considered a hallmark morphological feature of apoptosis but not necrosis?
(A) Progressive swelling of the cell body and organelles
(B) Condensed and fragmented chromatin with intact membranes
(C) Massive inflammatory responses in surrounding tissues
(D) Acute, enzymatic rupture of the outer plasma membrane

2021

189. A malignant tumor arising from the stratified squamous epithelium of the skin is formally called:
(A) Squamous cell carcinoma
(B) Squamous cell papilloma
(C) Basal cell carcinoma
(D) Epidermoid sarcoma

2018

190. Which of the following defines the term "carcinoma in situ"?
(A) A benign glandular neoplasm displaying purely epithelial origin
(B) A malignant tumor that has actively invaded the lymphatic system
(C) A malignant epithelial tumor that has not breached the membrane
(D) A mesenchymal tumor remaining strictly confined to its site of origin

2007

191. A tumor that is grossly encapsulated, grows slowly by expansion, and remains localized is generally considered:
(A) Malignant
(B) Metastatic
(C) Anaplastic
(D) Benign

2008

192. Which genetic element is a normal host gene that, when mutated or overexpressed, drives unregulated cell growth?
(A) Tumor suppressor gene
(B) Viral oncogene
(C) Proto-oncogene
(D) Apoptotic gene

2010

193. The normal function of the Rb (Retinoblastoma) gene product is to:
(A) Stimulate the physiological transition from G1 to S phase
(B) Inhibit the E_2F factor to block cell cycle progression
(C) Phosphorylate specific regulatory proteins during active mitosis
(D) Actively repair double-stranded DNA structural breaks

2018

194. The multi-hit hypothesis of carcinogenesis implies that:
(A) A single somatic mutation is always sufficient to induce cancer
(B) Direct physical trauma to a tissue directly initiates cancer
(C) Both cellular alleles of a proto-oncogene must be inactivated
(D) Cancer develops from multiple genetic mutations over time

NODIA

2008

195. Which type of RNA retrovirus requires integration near a host proto-oncogene to cause cancer via insertional mutagenesis, typically involving a long latent period?
(A) Slow-transforming retroviruses
(B) Acute-transforming retroviruses
(C) Cytolytic viruses
(D) DNA poxviruses

2013

196. In the multi-step model of chemical carcinogenesis, a "promoter" is defined as an agent that:
(A) Directly damages cellular DNA to introduce a permanent genetic mutation
(B) Induces clonal expansion and proliferation of a previously initiated cell
(C) Chemically neutralizes mutagenic compounds before they can enter the nucleus
(D) Reverses the malignant phenotype of an established tumor cell line

2004

197. Which molecular diagnostic technique is used to distinguish a monoclonal population of lymphocytes (diagnostic for lymphoma) from a

ANSWERS

1. (A) An increase in cell size leading to an increase in organ size
Cellular hypertrophy is an adaptive response characterized by an increase in the size of individual cells, which subsequently leads to an increase in the overall size of the organ. This change occurs without cell division and is typically seen in permanent cell populations like striated muscle cells when they are subjected to increased work demands.

2. (C) Increased synthesis of proteins and organelles
Physiological hypertrophy in permanent, post-mitotic cells such as striated muscle fibers is primarily driven by an increased rate of protein synthesis and the biogenesis of cellular organelles. This cellular mechanism allows the myofibrils to grow in size and strength, enhancing their structural and functional capacity to meet increased mechanical and workload demands placed upon them.

3. (A) Tissues containing cell populations capable of replication
Hyperplasia is an adaptive increase in the number of cells within an organ or tissue. It can only occur in cell populations that retain the capacity to divide, such as labile or stable cells. Tissues composed of permanent cells cannot undergo this adaptation because they lack the mitotic capability required to produce new cellular units.

4. (D) Regeneration of liver tissue after partial hepatectomy
Compensatory hyperplasia is a physiological adaptation where remaining tissue proliferates to restore function after loss or damage. A classic example is liver regeneration following a partial hepatectomy, where hepatocytes exit their resting state and undergo mitotic division to restore the original mass of the organ, driven by various growth factors and cytokine signaling pathways.

5. (A) P-2, Q-1, R-4, S-3
Organ development failures and adaptations are categorized by specific definitions. Agenesis represents the complete absence of an organ and its primordium, whereas aplasia indicates a failure to develop beyond a rudimentary structure. Hypoplasia refers to underdevelopment with fewer cells, and atrophy is the shrinkage of an organ that was once normally developed and functional.

6. (C) The kidney failed to form due to absence of the ureteric bud
Unilateral renal agenesis represents the complete congenital absence of one kidney, which occurs due to a failure in embryogenesis. This developmental defect typically arises when there is a complete absence of the ureteric bud or its failure to interact with the metanephric blastema, preventing the initiation of organ development and primordium formation.

7. (D) Aplasia has a rudimentary tissue mass, whereas agenesis lacks a primordium
The primary morphological distinction between these two developmental failures lies in the presence of primordial structures. In agenesis, there is a complete lack of any organ formation, including its primordium. In contrast, aplasia features a failure of the organ to develop properly, but a rudimentary, non-functional tissue mass or primordium remains present at the anatomical site.

8. (A) Viral destruction of mitotic cells during organogenesis
Feline panleukopenia virus targets rapidly dividing cells in the host body. When a pregnant queen is infected, the virus crosses the placenta and infects the fetus, selectively destroying the actively mitotic external germinal layer cells in the developing cerebellum during late gestation, which leads directly to severe cerebellar hypoplasia in the newborn kittens.

9. (B) Segmental underdevelopment of the intestine
Localized tissue hypoplasia refers to the incomplete development of a specific, circumscribed part of an organ or anatomical structure, resulting in a reduced cell number and size. Segmental underdevelopment of the intestine is a classic congenital example where only a specific section of the bowel fails to grow

Downloaded from www.pdf.tube

to its normal physiological size during embryonic development.

10. (C) A is true but R is false.
Disuse atrophy of skeletal muscle occurs when workload is decreased, which is a highly reversible process once normal physical activity or mechanical load is resumed. This recovery is possible because the atrophic cells are not dead; they have merely shrunk in size by degrading internal proteins and organelles to survive the period of reduced demand.

11. (A) The ubiquitin-proteasome pathway
Cellular protein degradation during tissue atrophy is primarily mediated by the ubiquitin-proteasome pathway. In this regulatory system, targeted proteins are first conjugated to ubiquitin molecules, which flags them for subsequent recognition and rapid enzymatic degradation within the large proteasome complex, leading directly to a marked decrease in total cellular mass and structural components.

12. (C) The lower motor neuron or its peripheral axon
Denervation atrophy is a rapid and severe form of muscle wasting that occurs following damage to the lower motor neuron or its peripheral axon. Because skeletal muscle fibers depend on continuous, low-level trophic signaling and electrical stimulation from these specific nerves to maintain their structural integrity, losing this input leads directly to cell shrinkage.

13. (D) Gradual reduction of arterial blood supply
Ischemic atrophy is caused by a gradual, progressive reduction in the arterial blood supply to a tissue or organ. This chronic deficiency in oxygen and essential nutrients forces the affected parenchymal cells to shrink in size and scale down their metabolic activities as a survival mechanism, rather than undergoing acute cell death or necrosis.

14. (C) Widening of sulci and narrowing of gyri
Senile atrophy of the brain, typically associated with normal aging and reduced blood supply from atherosclerosis, is characterized morphologically by the loss of cortical neurons. This structural loss physically manifests as a widening of the sulci and a corresponding narrowing of the gyri, particularly in the frontal and temporal lobes of the cerebral cortex.

15. (C) I and III
Endocrine atrophy occurs when a target organ loses vital trophic hormonal stimulation. For example, castration eliminates circulating testicular androgens, leading directly to the atrophy of the prostate gland. Conversely, losing adrenocorticotropic hormone from the pituitary results in progressive atrophy, not hypertrophy, of the adrenal cortex due to the absence of essential growth signals.

16. (D) Pathologic hyperplasia ceases when the stimulus is removed
Pathologic hyperplasia is a controlled, adaptive cellular proliferation driven by excessive hormonal stimulation or growth factors. Unlike neoplastic growth, which is fully autonomous and persists indefinitely, pathologic hyperplasia remains highly responsive to normal regulatory control mechanisms and will completely cease once the initiating hormonal or chemical stimulus is successfully removed from the tissue.

17. (A) Progesterone
Cystic endometrial hyperplasia in canines is most commonly driven by the excessive and prolonged influence of progesterone on an estrogen-primed uterus. This hormonal imbalance typically occurs during the luteal phase of the estrous cycle, leading to abnormal glandular secretion and proliferation, which frequently predisposes the affected uterus to secondary bacterial infections like pyometra.

NODIA

18. (C) Thyroid gland
Goiter represents a classic example of pathologic hyperplasia occurring specifically in the thyroid gland. This condition involves the non-neoplastic, diffuse enlargement of the thyroid tissue, which is driven by elevated circulating levels of thyroid-stimulating hormone in response to a deficiency in thyroid hormone production, resulting in the proliferation of follicular epithelial cells.

19. (A) Iodine
A chronic dietary deficiency of iodine is classically associated with the development of hyperplastic goiter. Iodine is an essential building block for thyroid hormone synthesis; its absence prevents the production of active hormones, triggering continuous feedback secretion of thyroid-stimulating hormone by the pituitary, which drives the sustained hyperplasia of the thyroid follicular cells.

20. (B) Priming by cytokines → Growth factor stimulation → Cell cycle entry → Proliferation
Liver regeneration through compensatory hyperplasia follows a highly coordinated pathway. Initially, hepatocytes are primed by inflammatory cytokines like interleukin-six to respond to stimuli. Next, growth factors such as hepatocyte growth factor stimulate these primed cells, prompting their entry into the cell cycle and driving active mitotic proliferation to successfully rebuild the lost organ mass.

21. (B) Metaplasia is a change to another mature cell type, while dysplasia is disordered cell growth
Metaplasia and dysplasia represent distinct pathological tissue changes. Metaplasia is a reversible, adaptive transformation where one fully mature, differentiated cell type is replaced by another mature cell type better suited to withstand environmental stress. Dysplasia, however, is characterized by disordered cellular growth, manifesting as a loss of architectural tissue orientation and cell uniformity.

22. (C) Vitamin A deficiency
Vitamin A is essential for maintaining the normal differentiation of epithelial tissues throughout the animal body. In avians, a deficiency in this vitamin leads directly to squamous metaplasia of the mucous-secreting columnar and cuboidal epithelium in the respiratory tract and esophageal glands, replacing it with a stratified squamous keratinizing epithelium that lacks defensive cilia.

23. (D) Reprogramming of stem cells to differentiate along a new mature pathway
Metaplasia does not occur from the direct phenotypic transformation of already mature, fully differentiated cells. Instead, it is driven by the genetic reprogramming of local tissue stem cells or undifferentiated mesenchymal cells. This changes their differentiation pathway, causing them to mature into a different cell type that is better adapted to withstand chronic irritation.

24. (A) Loss of architectural orientation and cell uniformity
Epithelial dysplasia is morphologically characterized by a significant loss of normal architectural orientation and cellular uniformity within the tissue. Under microscopic examination, dysplastic cells exhibit pronounced variation in size and shape, hyperchromatic nuclei, increased atypical mitotic figures, and a disorganized arrangement that disrupts the normal layered stratification of the epithelial membrane.

25. (A) P-1, Q-2, R-3, S-4
Metaplastic changes represent adaptive responses matching specific environmental stressors. Respiratory epithelium undergoes squamous metaplasia due to chronic cigarette smoke irritation. Soft tissue connective cells develop bone under traumatic influence. Avian esophageal glands show squamous changes from vitamin A deficiency, and extramedullary hematopoiesis arises in organs like the spleen when severe chronic anemia compromises marrow function.

26. (B) Formation of bone within an area of chronic muscle injury
Mesenchymal metaplasia specifically involves the adaptive transformation of one mature, differentiated connective tissue cell type into another. The formation of mature bone within skeletal muscle following chronic tissue injury, known as myositis ossificans, is a classic example of this process, where fibroblasts reprogram and differentiate into bone-producing osteoblasts instead of fibrous scar tissue.

27. (A) It has genetic and morphologic alterations that can progress to carcinoma
Dysplasia is considered a precancerous lesion because it exhibits significant genetic mutations and morphologic alterations that share structural characteristics with malignant cells. Although dysplastic tissue remains non-invasive and does not breach the basement membrane, the progressive accumulation of further genetic damage can drive its transition into an in-situ or invasive carcinoma over time.

28. (C) -oma
In medical oncology, tumor nomenclature is highly standardized to reflect cellular origin and behavior. The suffix "-oma" is appended to the specific tissue of origin to designate a benign neoplasm arising from mesenchymal cells, such as a fibroma for fibrous tissue or an osteoma for bone, representing localized, non-invasive growths of differentiated cells.

29. (D) Adenocarcinoma
Neoplasms are classified according to their tissue of origin and biological behavior. A malignant tumor that arises from glandular

Click Here to Buy book on Flipkart

highly tortuous, leaky, dilated, and structurally incomplete under microscopic evaluation.

87. (D) Telomeres
Cancer cells achieve replicative immortality primarily by avoiding the progressive, enzyme-induced shortening of telomeres, which are the protective DNA caps at the ends of chromosomes. In normal cells, telomeres shorten with each division, leading to senescence. Tumor cells prevent this by reactivating the enzyme telomerase, maintaining telomere length and enabling indefinite, continuous cellular replication.

88. (A) Inhibiting intrinsic apoptosis
The Bcl-two protein contributes to oncogenesis by directly inhibiting the intrinsic apoptotic pathway. As a key outer mitochondrial membrane protein, it prevents the release of cytochrome c into the cytoplasm by blocking the pro-apoptotic proteins Bax and Bak. This cellular mechanism prevents apoptosis, allowing mutated or damaged neoplastic cells to survive and proliferate continuously.

89. (B) HIF-1a transcription factor
As a solid tumor rapidly expands, its central core becomes highly hypoxic due to distance from existing capillaries. This lack of oxygen stabilizes Hypoxia-Inducible Factor one-alpha, preventing its normal degradation. HIF-one-alpha then translocates to the nucleus to bind target genes, directly triggering transcription of angiogenic factors like VEGF to promote blood vessel formation.

90. (D) A is false but R is true.
The assertion is false because tumor-associated macrophages do not always destroy tumor cells; they are frequently co-opted to actively promote tumor growth, matrix remodeling, and immunosuppression. However, the reason is true: these macrophages are recruited from circulating blood monocytes that migrate into the tumor microenvironment in response to chemokines secreted by neoplastic cells.

91. (A) Aerobic glycolysis
The Warburg effect describes a distinctive metabolic reprogramming in cancer cells. Even in the presence of abundant oxygen, neoplastic cells shift their active metabolism away from oxidative phosphorylation, relying heavily on aerobic glycolysis. This process converts glucose to lactate, providing the rapid energy and carbon skeletons required for continuous cellular component synthesis and division.

NODIA

92. (C) Lymphatic vessels
Carcinomas, which are malignant neoplasms of epithelial cell origin, most characteristically favor lymphatic vessels for their initial route of metastatic dissemination. Because epithelial organs are rich in lymphatics, detached tumor cells easily penetrate the thin walls of lymphatic capillaries, traveling to regional lymph nodes where they lodge in the subcapsular sinus and proliferate.

93. (D) Hematogenous spread
Sarcomas, which are malignant tumors of mesenchymal origin, typically favor the hematogenous route for their initial metastatic dissemination. Because mesenchymal tissues are highly vascularized, detached sarcoma cells actively invade local veins rather than lymphatics, traveling directly through systemic venous circulation to lodge in capillary beds of distant organs such as the lungs.

94. (C) The first node receiving lymph flow from the primary tumor
The sentinel lymph node represents a vital concept in clinical oncology and surgical staging. It is defined as the first lymph node in a regional lymphatic basin that receives drainage directly from the primary tumor. Identifying and biopsy of this specific node allows clinicians to accurately assess whether metastatic dissemination has initiated, avoiding unnecessary removal of other nodes.

95. (A) I, II and IV
Transcoelomic seeding is a major metastatic pathway that occurs when a malignant tumor penetrates the surface of an organ and sheds cells into a natural body cavity. It is a common route for ovarian carcinomas spreading within the peritoneal cavity, and frequently leads to neoplastic effusions, as represented by statements one, two, and four.

96. (D) Liver
Hematogenous spread of malignant tumors originating in the gastrointestinal tract typically results in secondary metastatic deposits in the liver. This specific metastatic pattern occurs

39. (D) Expansive growth, compressing surrounding tissue
Benign tumors are characterized by a slow, localized, and strictly expansive pattern of growth. As they slowly expand, they typically compress the surrounding normal tissues and frequently stimulate the formation of a protective, outer fibrous capsule. This structural containment keeps the tumor strictly localized, preventing infiltrative tissue destruction and any possibility of metastatic spread.

40. (A) Hyperplasia → Dysplasia → Carcinoma in situ → Invasive carcinoma
The standard sequence of progressive cellular changes during carcinogenesis begins with hyperplasia, characterized by an adaptive increase in cell number. Under persistent stress, this can progress to dysplasia, where cells exhibit abnormal development. Next, the tissue enters the stage of carcinoma in situ, before finally breaching the basement membrane to become an invasive carcinoma.

41. (B) Variation in cellular and nuclear size and shape
Cellular pleomorphism is a primary morphological hallmark of malignant neoplasms and refers specifically to a marked variation in cellular and nuclear size and shape. Unlike normal cells, which maintain uniform dimensions, malignant cells completely lose regulatory control of their cytoskeleton and cell cycle, leading to highly variable, disorganized, and abnormal structural appearances.

42. (D) 1:1
In normal, mature somatic cells, the nuclear-to-cytoplasmic volume ratio is relatively low, typically ranging from one-to-four to one-to-six. In contrast, malignant tumor cells undergo progressive nuclear enlargement and cytoplasmic reduction as they rapidly divide, shifting this critical ratio significantly towards one-to-one, reflecting highly active genetic material and increased cellular replication.

43. (A) Increased DNA content and clumped chromatin
Nuclear hyperchromasia is a common microscopic finding in malignant cells, characterized by intensely dark staining of the nucleus. This feature is primarily caused by an abnormally high content of genomic DNA and dense clumping of chromatin. These alterations reflect the high transcriptional activity, aneuploidy, and rapid replication cycles typical of neoplastic cellular populations.

44. (C) Malignant genomic instability
Atypical mitotic figures, such as tripolar, quadripolar, or multipolar spindles, represent highly abnormal cell division processes. Unlike normal bipolar mitoses seen in physiological regeneration, these bizarre configurations indicate severe damage to the mitotic spindle apparatus and a profound state of malignant genomic instability, leading directly to abnormal chromosomal segregation and aneuploidy in daughter cells.

45. (A) P-1, Q-2, R-3, S-4
Malignant cellular hallmarks are defined by specific microscopic alterations. Anaplasia corresponds to a complete lack of differentiation, while pleomorphism is characterized by variation in cell size and shape. Hyperchromasia is characterized by darkly stained nuclei from increased DNA, and atypical mitoses represent abnormal chromosomal segregation during division, such as tripolar or quadripolar spindle formations.

NODIA

46. (C) Elaboration of fetal proteins such as alpha-fetoprotein
Anaplastic tumor cells undergo functional derangement as they lose normal cellular differentiation. They often regress to embryonic phenotypes, leading to the abnormal synthesis and secretion of oncofetal proteins, such as alpha-fetoprotein or carcinoembryonic antigen, which are normally produced only during fetal development. These proteins serve as valuable diagnostic and monitoring biomarkers in clinical oncology.

47. (B) Initiation and promotion
Chemical carcinogenesis is a complex multi-step process classically divided into two major functional stages. The first stage is initiation, which involves rapid, irreversible DNA damage. This is followed by promotion, a step that does not alter DNA but stimulates the selective, clonal expansion of the initiated cells, leading to tumor formation over time.

48. (D) It induces permanent, irreversible DNA mutations
An initiating chemical carcinogen acts directly on the target cell genome to induce permanent, irreversible mutations in critical genes like proto-oncogenes or tumor suppressors. This cellular alteration is rapid and is permanently transmitted to all progeny cells, rendering them susceptible to subsequent promotion, even if a long interval occurs before exposure to the promoting agent.

49. (A) Are electrophiles that do not require metabolic conversion
Direct-acting chemical carcinogens are highly reactive, electron-deficient electrophiles that directly attack and bind to cellular DNA, RNA, and proteins without requiring any prior metabolic activation or conversion. This distinguishes them from indirect-acting carcinogens, or procarcinogens, which must be enzymatically converted by host metabolic pathways, particularly cytochrome P-450, to become active, DNA-damaging electrophiles.

50. (D) A is false but R is true.
The assertion is false because tumor promoters do not induce direct DNA damage or cause strand breaks. Instead, the reason is entirely true: promoters act through epigenetic pathways to stimulate cellular proliferation and drive the clonal expansion of previously initiated cells. This sustained growth increases the likelihood of acquiring secondary mutations that lead to malignancy.

51. (A) Hepatocellular carcinoma
Aflatoxin B1 is a potent, naturally occurring mycotoxin produced by Aspergillus fungi. As an indirect carcinogen, it undergoes metabolic activation in the liver by cytochrome P-450 enzymes to form a highly reactive epoxide. This active metabolite binds to DNA, causing a specific mutation in the p53 tumor suppressor gene that drives hepatocellular carcinoma.

52. (D) Polycyclic aromatic hydrocarbons
Polycyclic aromatic hydrocarbons represent a major class of potent chemical carcinogens. These compounds are formed during the incomplete combustion of organic material and are found in high concentrations in tobacco smoke, exhaust fumes, and smoked or grilled meats. After metabolic activation, they form reactive diol epoxides that bind covalently to DNA, initiating carcinogenesis.

53. (B) Causes mutations in bacteria
The Ames test is a rapid, in vitro biological assay designed to assess the mutagenic potential of chemical compounds. Using a mutant strain of Salmonella typhimurium that cannot synthesize histidine, the test measures the chemical's ability to induce reverse mutations that restore histidine production. Since mutagenicity correlates with carcinogenicity, it serves as an effective screening tool.

54. (C) Leukemia or lymphoma
Alkylating agents like cyclophosphamide are potent chemotherapeutic drugs that kill cancer cells by forming cross-links in DNA. However, because they are also mutagenic, prolonged administration carries a significant long-term risk of inducing secondary malignancies. These secondary neoplasms most commonly manifest as acute myeloid leukemia or lymphoma due to therapy-induced damage to hematopoietic stem cells.

55. (A) II, III and V
In chemical carcinogenesis, initiators cause rapid, irreversible DNA damage, whereas promoters do not mutate DNA but stimulate reversible cell division, which is critical for clonal expansion. Procarcinogens are typically metabolized and activated in the liver. These processes are represented by statements two, three, and five, while statement one is incorrect because initiation must precede promotion.

56. (A) Pyrimidine dimers
Ultraviolet radiation primarily damages cellular DNA through a photochemical reaction that induces the formation of covalent cross-links between adjacent pyrimidine bases, particularly thymine or cytosine. This results in cyclobutane pyrimidine dimers, which physically distort the DNA double helix, leading to transcription errors and replication blocks that can introduce permanent mutations if left unrepaired.

57. (B) UVB
Ultraviolet B (UVB) radiation, spanning wavelengths of 280 to 320 nanometers, is the most potent inducer of skin cancers like squamous cell carcinoma and cutaneous melanoma. Unlike UVA, which primarily acts

Downloaded from www.pdf.tube

indirectly by generating free radicals, UVB is directly absorbed by cellular DNA, efficiently causing the formation of pyrimidine dimers and subsequent mutations in key regulatory genes.

58. (B) Generation of ROS and DNA double-strand breaks
Ionizing radiation promotes carcinogenesis through both indirect and direct mechanisms. It radiolyzes intracellular water molecules, generating highly reactive oxygen species (ROS) that chemically damage DNA. Additionally, it possesses enough energy to directly break the phosphodiester backbone of DNA, causing double-strand breaks that are difficult to repair, often leading to genomic rearrangements, translocations, and mutations.

59. (A) Prolonged ultraviolet radiation
Bovine ocular squamous cell carcinoma, commonly known as cancer eye, is highly correlated with prolonged exposure to solar ultraviolet radiation. This condition is particularly prevalent in breeds like Hereford cattle that lack periocular pigmentation, where the absence of melanin allows UV rays to directly damage cellular DNA in the corneal epithelium, initiating tumorigenesis.

60. (B) UV exposure → Thymine dimer formation → Failure of nucleotide excision repair → Mutation in oncogene → Carcinoma
Skin carcinogenesis induced by ultraviolet radiation follows a specific, step-by-step path. First, UV exposure occurs, which directly triggers thymine dimer formation within the DNA. If nucleotide excision repair systems fail to fix these dimers, DNA replication proceeds with errors, leading to mutations in oncogenes or tumor suppressors, ultimately driving the development of cutaneous carcinoma.

61. (D) Thyroid carcinoma
Exposure to radioactive isotopes of iodine, such as iodine-131 released during nuclear accidents or therapy, increases the risk of thyroid carcinoma. Because the thyroid gland actively concentrates iodine from the bloodstream to synthesize thyroid hormones, any ingested or inhaled radioactive iodine selectively accumulates in thyroid tissue, exposing follicular cells to localized, damaging radiation.

62. (C) Xeroderma pigmentosum
Xeroderma pigmentosum is a rare human recessive genetic disorder that serves as a classic model for defective DNA repair. Patients with this condition have inherited mutations in genes essential for nucleotide excision repair, leaving them unable to repair pyrimidine dimers caused by ultraviolet light, leading to extreme sun sensitivity and high incidence of skin cancers.

63. (B) Lung cancer
Chronic exposure to radon gas, a naturally occurring radioactive gas formed by the decay of uranium in soil and rock, is a well-documented risk factor for lung cancer. Inhaled radon decay products emit high-energy alpha particles that directly damage the DNA of respiratory epithelial cells, inducing mutations that initiate and promote bronchogenic carcinoma.

64. (D) Chromosomal translocations and deletions
At the cellular level, the primary danger of ionizing radiation is its high energy, which can induce severe double-strand breaks in DNA. When the cell attempts to repair these breaks, improper joining of chromosomal ends frequently occurs, leading to structural changes like chromosomal translocations and deletions that can activate oncogenes or silence tumor suppressor genes.

NODIA

65. (A) P-3, Q-1, R-2, S-4
Oncogenic viruses are linked to specific neoplasias across animal species. Papillomaviruses cause cutaneous warts and squamous carcinomas. Gallid herpesvirus two causes Marek's disease, a T-cell lymphoma in poultry. Bovine leukemia virus induces enzootic bovine leukosis in cattle, and feline leukemia virus is a major driver of mediastinal lymphoma and other hematopoietic malignancies in cats.

66. (C) Tumor suppressor proteins like p53 and Rb
Oncogenic DNA viruses promote tumorigenesis primarily by expressing specific viral oncoproteins that bind to and inactivate host tumor suppressor proteins, most notably p53 and the retinoblastoma protein. By neutralizing these master cell cycle regulators, the virus bypasses critical checkpoints, allowing infected host cells to divide uncontrollably and accumulate genomic mutations, driving neoplastic transformation.

epithelial cells or exhibits glandular growth patterns under microscopic examination is termed an adenocarcinoma. This contrasts with an adenoma, which is benign, or sarcomas and carcinomas, which arise from mesenchymal or non-glandular epithelial tissues respectively.

30. (C) A is true but R is false.
Lymphoma is a highly malignant neoplasm composed of neoplastic lymphoid cells, making the first statement true. However, the second statement is false because there are several critical exceptions in oncological nomenclature where the suffix "-oma" designates a highly malignant tumor rather than a benign one, such as lymphoma, melanoma, seminoma, and mesothelioma.

31. (B) Melanoma
While the suffix "-oma" typically indicates a benign neoplasm, melanoma represents a classic and critical exception in oncological nomenclature. It is a highly aggressive and malignant tumor that arises from melanocytes, which are the pigment-producing cells of the skin and eye. It is characterized by high metastatic potential, requiring rapid clinical identification and therapeutic intervention.

32. (D) Leiomyoma
Smooth muscle tumors are classified based on their biological behavior. A benign neoplasm composed entirely of smooth muscle cells is termed a leiomyoma, commonly occurring in the uterus or gastrointestinal tract. A malignant smooth muscle tumor is a leiomyosarcoma, whereas benign and malignant skeletal muscle tumors are termed rhabdomyomas and rhabdomyosarcomas, respectively.

33. (A) It contains cells from more than one germ layer, often all three
A teratoma is a highly unique neoplasm composed of various tissues derived from more than one embryological germ layer, and frequently contains elements of all three: ectoderm, mesoderm, and endoderm. These tumors typically arise from totipotent germ cells in the gonads and may contain highly recognizable structures like hair, teeth, bone, and neural tissue.

34. (C) Hamartoma
A hamartoma is a benign, non-neoplastic developmental malformation characterized by a disorganized mass of mature specialized cells and tissues that are completely indigenous to the specific anatomical site where they occur. Although histologically abnormal in structural arrangement, the cells are fully mature and lack the capability for autonomous, uncontrolled neoplastic expansion.

35. (B) II and IV
Mesenchymal tumors follow highly specific naming and metastatic spreading rules. A malignant cartilage tumor is a chondrosarcoma, not an osteosarcoma, which originates in bone cells. Additionally, sarcomas typically metastasize early via the hematogenous route rather than through lymphatics. This makes the second and fourth statements incorrect, while the other statements correctly define mesenchymal tumors.

36. (D) Ability to invade local tissues and metastasize
The ultimate and most reliable distinguishing hallmark of a malignant neoplasm compared to a benign tumor is its ability to invade adjacent normal tissues and metastasize to distant anatomical sites. While benign tumors grow slowly and remain localized within a fibrous capsule, malignant tumors exhibit infiltrative borders that compromise and destroy surrounding tissues.

37. (B) The extent to which neoplastic cells resemble normal parenchymal cells
In clinical oncology, cellular differentiation describes the exact degree to which neoplastic cells morphologically and functionally resemble their normal, healthy cells of origin. Well-differentiated tumors closely mimic mature host tissues and are typical of benign growths, whereas poorly differentiated or undifferentiated tumors show total loss of structural features, which is highly associated with malignancy.

38. (C) Complete lack of differentiation
Anaplasia refers specifically to a complete lack of structural and functional cellular differentiation. Anaplastic tumor cells have retrogressed to a primitive, embryonic-like state and no longer resemble their tissue of origin. This feature is a definitive morphologic hallmark of highly aggressive malignancies, characterized by marked pleomorphism, hyperchromatic nuclei, and frequent atypical mitotic figures.

Click Here to Buy book on Amazon

77. (D) A gene that inhibits cell growth or promotes apoptosis
A tumor suppressor gene is a normal cellular gene whose primary function is to negatively regulate cell division, or to trigger apoptosis under conditions of severe genetic damage. These genes act as critical brakes on cellular proliferation. The functional inactivation of both alleles of a tumor suppressor gene allows cells to divide uncontrollably, promoting tumorigenesis.

78. (C) G1 to S phase
The Retinoblastoma protein is a key regulator of the cell cycle, controlling the transition from the G1 to the S phase. In its active, hypophosphorylated state, Rb binds and silences the E_2F transcription factor. When stimulated, cyclin-dependent kinases phosphorylate Rb, releasing E_2F and allowing the transcription of genes required for DNA replication.

79. (B) Arrest the cell cycle for DNA repair or trigger apoptosis
The p53 gene is known as the guardian of the genome due to its role in maintaining genetic integrity. When DNA damage occurs, p53 accumulates and activates the transcription of genes that arrest the cell cycle to allow for repair. If the damage is beyond repair, p53 triggers apoptosis, preventing propagation of mutated, oncogenic cells.

80. (B) DNA damage → p53 accumulation → p21 transcription → Cell cycle arrest
Under physiological conditions, cellular exposure to genotoxic stress initiates a coordinated sequence of regulatory events. First, DNA damage is detected, which immediately triggers the stabilization and accumulation of the p53 protein. This accumulated p53 drives the transcription of the p21 gene, a kinase inhibitor, which physically blocks cell cycle progression, leading directly to arrest.

81. (A) Tumor suppressor genes
Loss of heterozygosity is a genetic alteration highly relevant to the functional inactivation of tumor suppressor genes. Since these genes behave recessively, a patient carrying one mutated germline allele remains healthy. However, if the remaining wild-type allele is lost through deletion or somatic mutation, the cell completely loses tumor suppressive function, initiating neoplasia.

82. (C) Chromosomal translocation
The c-myc proto-oncogene is activated in Burkitt lymphoma through a classic chromosomal translocation. This structural mutation typically swaps segments of chromosomes eight and fourteen, placing c-myc under the control of the active immunoglobulin heavy-chain promoter. This results in the continuous, unregulated overproduction of the Myc transcription factor, driving rapid, malignant B-cell proliferation.

83. (A) 1-2 millimeters
Diffusion of oxygen and vital nutrients from existing blood vessels is physically limited to a distance of approximately one to two millimeters. Consequently, any growing solid tumor must initiate and stimulate its own blood vessel formation, a process known as tumor angiogenesis, to exceed this size, receive nutrition, and successfully survive or expand further.

84. (D) Vascular Endothelial Growth Factor
Vascular Endothelial Growth Factor is the most critical pro-angiogenic molecule secreted by tumor cells to stimulate new blood vessel growth. When a solid tumor experiences internal hypoxia, it upregulates VEGF production. This protein binds to tyrosine kinase receptors on nearby endothelial cells, driving their proliferation, migration, and assembly into new, functional tumor-supplying capillaries.

NODIA

85. (A) P-2, Q-1, R-3, S-4
Tumors utilize distinct cellular mechanisms to promote growth and survival. Angiogenesis is driven by VEGF secretion to recruit new capillaries. Replicative immortality is achieved through telomerase reactivation, which maintains chromosome ends. Apoptosis is evaded by overexpressing anti-apoptotic Bcl-two, and immune evasion is facilitated by downregulating host MHC class-one molecules to escape cytotoxic T-cell detection.

86. (C) They are tortuous, leaky, and structurally incomplete
Newly formed tumor blood vessels display significant structural abnormalities compared to normal physiological vasculature. Because they are produced rapidly under the disorganized influence of excessive pro-angiogenic factors like VEGF, these vessel walls lack a proper muscular layer and basement membrane. This leaves them

67. (A) p53 protein
The E6 oncoprotein of high-risk human papillomaviruses contributes to carcinogenesis by binding to the host p53 tumor suppressor protein. This binding recruits the cellular ubiquitin ligase E6AP, targeting p53 for rapid degradation via the proteasome. Consequently, the host cell loses its ability to arrest the cell cycle or undergo apoptosis in response to DNA damage.

68. (D) T lymphocytes
Gallid herpesvirus two, the causative agent of Marek's disease in chickens, is an oncogenic alphaherpesvirus that specifically targets T lymphocytes. The virus infects these cells and expresses viral oncogenes, such as the meq gene, which mimics host bZIP transcription factors, leading to the rapid, malignant transformation of T cells and subsequent multi-organ lymphoma development.

69. (B) Insertional mutagenesis near a host proto-oncogene
Retroviruses like feline leukemia virus typically lack their own viral oncogenes, but can cause cancer through insertional mutagenesis. Upon entering the host cell, the viral reverse transcriptase generates a DNA copy that integrates randomly into the host genome. If integration occurs near a host proto-oncogene, the strong viral promoters drive its overexpression, initiating malignant transformation.

70. (D) A is false but R is true.
The assertion is false because RNA tumor retroviruses do not integrate directly into host RNA; instead, they must first synthesize a DNA intermediate. This mechanism is correctly described in the reason, which is true: retroviruses use their reverse transcriptase enzyme to create a proviral DNA copy that integrates directly into the host cell genome.

71. (D) B lymphocytes
Bovine leukemia virus is an exogenous, oncogenic retrovirus that is the causative agent of enzootic bovine leukosis in cattle. The virus specifically targets and infects B lymphocytes, leading to a state of persistent lymphocytosis and, in a percentage of infected animals, the development of malignant B-cell lymphosarcomas in various organs and lymph nodes.

72. (A) tax
The tax gene of the bovine leukemia virus encodes a highly specialized viral regulatory protein that functions as a transcriptional transactivator. Rather than acting as a direct cellular oncogene, the Tax protein activates the transcription of both viral genes and host genes involved in cell cycle progression, driving B-lymphocyte proliferation and promoting malignant transformation.

73. (B) Inducing severe systemic immunosuppression
Feline immunodeficiency virus is a lentivirus that does not carry its own viral oncogenes nor does it directly cause malignant transformation. Instead, it targets and depletes T-helper lymphocytes, inducing a state of severe systemic immunosuppression. This immunodeficient state impairs the host's tumor surveillance mechanisms, allowing opportunistic oncogenic viruses to proliferate and drive lymphoma development.

74. (B) Normal cellular genes promoting cell growth
Proto-oncogenes are normal, highly regulated cellular genes that play essential roles in promoting cell growth, proliferation, and survival under physiological conditions. They encode proteins such as growth factors, receptor tyrosine kinases, signal transducers, and transcription factors. When these genes undergo mutations or gain-of-function alterations, they become active oncogenes, driving uncontrolled, autonomous neoplastic growth.

75. (B) II only
The ras gene is a cellular proto-oncogene encoding a small G-protein essential for signal transduction. Normally, Ras cycles between active GTP-bound and inactive GDP-bound states. Mutated forms of Ras are oncogenic because they remain permanently bound to GTP and fail to hydrolyze it, sending continuous, uncontrolled growth signals to the nucleus.

76. (C) Nuclear transcription factor
The myc oncogene encodes a highly potent nuclear transcription factor that regulates the expression of genes essential for cell growth, protein synthesis, and cell cycle progression. Under physiological conditions, its expression is tightly controlled. When mutated or amplified in cancer, continuous Myc activity drives autonomous transcription, facilitating cell cycle entry and promoting cellular transformation.

Downloaded from www.pdf.tube

because venous blood draining from the stomach and intestines enters the portal vein, which flows directly into the liver, allowing circulating tumor cells to lodge in the hepatic sinusoidal capillaries.

97. (A) Lungs
In systemic hematogenous metastasis, tumor cells that invade veins draining the head, limbs, or body wall enter the systemic venous circulation. This blood travels through the vena cava directly to the right side of the heart, which pumps it into the pulmonary arteries. Consequently, these tumor emboli first lodge in the extensive capillary beds of the lungs.

98. (C) Abundant splenic immune cells and harsh hemodynamics
Although the spleen receives a massive volume of arterial blood, it is rarely a site for gross secondary tumor metastasis. This protection is attributed to the spleen's unique microenvironment, which contains an abundance of active immune cells like lymphocytes and macrophages, combined with harsh hemodynamics and physical filtration structures that actively destroy circulating tumor cells.

99. (A) A tumor bypasses regional lymph nodes to form metastases in more distant lymph nodes
In the lymphatic dissemination of cancer, tumor cells typically travel sequentially through regional lymphatic channels to the nearest draining nodes. However, skip metastasis refers to a highly clinical scenario where tumor cells bypass these expected regional lymph nodes entirely, moving deeper into the lymphatic system to establish metastatic growths in more distant lymph nodes.

100. (A) I and III
Lymphatic dissemination follows specific biological principles. Sarcomas, such as fibrosarcomas, favor hematogenous, not lymphatic, spread, making the first statement incorrect. Additionally, regional lymph node enlargement near a tumor does not always indicate metastasis; it can represent a reactive, hyperplastic immune response to tumor antigens, making the third statement incorrect. The other statements accurately describe lymphatic dissemination.

101. (B) Loss of E-cadherin mediated cell-cell adhesion
An essential initial requirement for a carcinoma to undergo local invasion is the loss of E-cadherin mediated cell-to-cell adhesion. Under physiological conditions, E-cadherins form tight intercellular junctions that anchor epithelial cells together. The downregulation or functional loss of these proteins allows neoplastic cells to detach from one another, initiating migration through the basement membrane.

102. (C) Secretion of Matrix Metalloproteinases (MMPs)
The degradation of the extracellular matrix and basement membrane by invading tumor cells is primarily facilitated by their secretion of Matrix Metalloproteinases. These zinc-dependent proteolytic enzymes actively cleave structural proteins like collagen and laminin within the basement membrane and interstitial connective tissues, creating a physical pathway that allows detached neoplastic cells to migrate locally.

NODIA

103. (D) Penetrate a vessel wall to enter circulation
Intravasation is a key sequential step in the metastatic cascade where tumor cells actively penetrate the basement membrane and endothelial layer of a blood or lymphatic vessel to enter the circulatory system. This active process requires cell motility, structural remodeling of the vessel wall, and the localized secretion of proteolytic enzymes to facilitate entry.

104. (A) Platelets and coagulation factors
Once inside the bloodstream, circulating tumor cells face harsh hemodynamics and host immune defenses, particularly natural killer cells. To survive, these tumor cells frequently aggregate with host platelets and coagulation factors to form protective micro-emboli. This cellular shield protects the tumor cells from physical shear stress and conceals them from active immune surveillance.

105. (A) I and II
In the metastatic cascade, intravasation, where cells enter the blood vessels, must precede extravasation, where they exit. During this process, circulating tumor cells must survive the immense physical shear stress of blood flow. However, only a tiny fraction of cells that enter circulation successfully survive to establish

internal complexity, and specific cell surface marker profiles.

144. (C) Exophytic, branching, and cauliflower-like growths
Papillomas are benign epithelial neoplasms that arise from squamous or transitional epithelium, presenting grossly with a characteristic exophytic, branching, or cauliflower-like growth pattern. Microscopically, these lesions are composed of finger-like projections of vascularized fibrous connective tissue cores covered by layers of proliferating, well-differentiated and benign epithelial cells.

145. (B) Keratin pearls
Squamous cell carcinomas are characterized histologically by the formation of keratin pearls, which are concentric, laminated nests of keratinizing squamous epithelial cells. These structures represent the neoplastic cells' attempt to differentiate, trapping keratin within the center of the cellular nests, and serve as a diagnostic hallmark of well-differentiated squamous cell malignancies.

146. (D) Abundant, dense fibrous connective tissue stroma
A scirrhous adenocarcinoma is characterized by a prominent desmoplastic reaction, which involves the production of abundant, dense fibrous connective tissue stroma surrounding the neoplastic cells. This reactive stromal proliferation is stimulated by tumor-derived growth factors, imparting a firm, hard consistency to the tumor on palpation and gross examination.

147. (A) Urinary bladder
Urothelial carcinomas, historically known as transitional cell carcinomas, arise from the specialized transitional epithelium lining the urinary tract. While they can occur anywhere along the urinary system, they are most frequently located in the urinary bladder, particularly in the trigone region, where they often cause hematuria and dysuria.

148. (C) Solitary, discrete, and benign nodules in the skin
Basal cell tumors in domestic animals typically present as solitary, discrete, and benign cutaneous nodules, most commonly found on the head, neck, or shoulders. Although these neoplasms arise from the basal layer of the epidermis or hair follicles, they exhibit very low metastatic potential and are usually cured by surgical excision.

149. (A) Adnexal glands of the skin
Sebaceous adenomas are common, benign glandular tumors arising from the adnexal sebaceous glands of the skin. They are frequently observed in older dogs, presenting as small, wart-like, or lobulated cutaneous masses. Microscopically, they are composed of lobules of well-differentiated sebocytes containing lipid droplets, surrounded by a basaloid germinal layer.

150. (D) Plump spindle cells arranged in interlacing herringbone patterns
Fibrosarcomas are malignant mesenchymal neoplasms histologically characterized by proliferating, plump spindle-shaped cells arranged in interlacing bundles that form classic herringbone patterns. These neoplastic cells display variable nuclear atypia, pleomorphism, and mitotic activity, and are associated with a variable collagenous matrix which they actively synthesize.

151. (B) Osteoid or unmineralized matrix
Osteosarcomas are highly aggressive, malignant neoplasms of bone characterized by the direct production of osteoid, which is an unmineralized bone matrix, by the neoplastic osteoblasts. This histological finding is pathognomonic, distinguishing osteosarcomas from other bone tumors, and is critical for confirming the diagnosis under routine microscopic evaluation.

152. (C) Lungs
Appendicular osteosarcomas in large and giant breed dogs are characterized by highly aggressive biological behavior, with early and widespread hematogenous metastasis. The lungs are the primary and most common site for secondary metastatic colonization, often containing micrometastases at the time of initial clinical presentation and diagnosis.

153. (D) Vascular endothelial cells
Hemangiosarcomas are highly malignant, vasoformative neoplasms that originate from vascular endothelial cells. Because of their endothelial origin, these aggressive tumors characteristically form irregular, blood-filled channels and spaces, displaying high rates of local

Click Here to Buy book on Flipkart

complex class I molecules, secretion of immunosuppressive cytokines like transforming growth factor-beta, expression of checkpoint molecules like programmed death-ligand 1, and recruitment of regulatory T cells. Upregulation of highly immunogenic antigens would instead promote immune recognition and clearance.

116. (C) It binds PD-1 on T cells, sending inhibitory signals that halt T cell attack
The expression of programmed death-ligand 1 on tumor cell surfaces acts as an immune checkpoint to evade host defenses. When this ligand binds to programmed death-1 receptors on cytotoxic T cells, it transmits inhibitory signals that suppress T-cell activation, induce functional exhaustion, and prevent the lymphocytes from destroying the neoplastic cells.

117. (A) Secreting growth factors, promoting angiogenesis, and suppressing immunity
Tumor-associated macrophages displaying the M2 activation phenotype actively facilitate tumor progression and metastasis. These specialized cells secrete pro-survival growth factors, promote neoangiogenesis by releasing vascular endothelial growth factor, remodel the extracellular matrix to assist invasion, and release anti-inflammatory cytokines that suppress antitumor immune responses within the local microenvironment.

118. (D) Dense desmoplastic stroma
A dense desmoplastic stroma acts as a formidable physical barrier that restricts the infiltration of immune cells into solid tumors. This fibrotic microenvironment, characterized by abundant collagen deposition and activated myofibroblasts, increases interstitial fluid pressure and forms a dense mechanical meshwork, effectively shielding neoplastic cells from cytotoxic lymphocytes and therapeutic agents.

119. (B) Cytotoxic Natural Killer (NK) cells
Antibody-dependent cellular cytotoxicity is an immune response primarily mediated by natural killer cells. These cytotoxic cells express surface Fc-gamma receptors that recognize and bind to the Fc region of antibodies attached to tumor-associated antigens. Upon binding, the natural killer cells undergo degranulation, releasing perforin and granzymes to destroy the targeted tumor cell.

120. (C) Systemic release of cytokines like TNF-alpha by the tumor and host
Cancer cachexia is a complex metabolic syndrome characterized by progressive loss of skeletal muscle and adipose tissue. This wasting is primarily driven by systemic inflammatory cytokines, notably tumor necrosis factor-alpha, interleukin-one, and interleukin-six, which are released by both the tumor and host immune cells, altering metabolic pathways and accelerating protein degradation.

121. (D) Inhibiting iron absorption and locking iron within macrophages
Hepcidin is a key regulatory hormone that mediates anemia of chronic disease in cancer patients by controlling systemic iron availability. Elevated hepcidin levels lead to the degradation of ferroportin, which prevents enterocytes from absorbing dietary iron and locks existing iron stores inside macrophages, restricting the iron required for active erythropoiesis.

122. (A) Are independent of local tumor growth, metastasis, or indigenous hormone production
Paraneoplastic syndromes are systemic symptom complexes that occur in cancer patients but cannot be directly attributed to the local invasion, metastasis, or normal anatomical hormone production of the primary tumor. Instead, they are triggered by tumor-derived bioactive substances, such as ectopic hormones, cytokines, or autoantibodies, affecting distant tissues and organ systems.

123. (B) Parathyroid Hormone-Related Protein (PTHrP)
Hypercalcemia of malignancy is a common endocrine paraneoplastic syndrome driven by the ectopic production of parathyroid hormone-related protein by neoplastic cells. This tumor-derived peptide mimics endogenous parathyroid hormone by binding to osteoblast receptors, which stimulates bone resorption and increases renal calcium reabsorption, leading to elevated systemic calcium concentrations.

124. (B) Lungs or thoracic cavity
Hypertrophic osteopathy is a paraneoplastic syndrome characterized by bilateral, symmetrical periosteal new bone formation along the distal extremities. This condition is most frequently associated with space-occupying lesions in the lungs or thoracic cavity, such as primary

Downloaded from www.pdf.tube

exhibit cellular pleomorphism, loss of polarity, and destructive infiltrative growth.

164. (C) Cellular pleomorphism, mitotic count, and invasiveness
The Patnaik and Kiupel grading systems for canine mast cell tumors are histopathological tools that evaluate microscopic features to predict biological behavior. These systems assess cellular pleomorphism, mitotic count per high-power fields, nuclear aberrations, multinucleation, and deep tissue invasiveness, providing critical prognostic guidance regarding the likelihood of metastasis and recurrence.

165. (A) The cells lack the characteristic melanin pigment
Amelanotic melanomas represent a challenging diagnostic entity because the neoplastic cells lack the characteristic brown-black melanin pigment normally found in melanocytic tumors. Under standard hematoxylin and eosin staining, these poorly differentiated cells can mimic other round cell tumors, sarcomas, or carcinomas, necessitating specialized diagnostic techniques to confirm their lineage.

166. (D) Melan-A or PNL2
Confirming the diagnosis of an amelanotic melanoma typically requires immunohistochemical staining for specific melanocytic lineage markers. Pathologists utilize antibodies against Melan-A, PNL2, or S100, which remain expressed within the cells despite the absence of visible melanin pigment, allowing for definitive differentiation from other poorly differentiated neoplasms.

167. (B) A robust host cell-mediated immune response targeting tumor cells
The spontaneous regression of canine transmissible venereal tumor is primarily mediated by a robust host cell-mediated immune response. Following an initial phase of progressive growth where the tumor evates detection, host T lymphocytes, particularly cytotoxic T cells, infiltrate the mass, secreting cytokines that trigger widespread apoptosis of the neoplastic allograft.

168. (C) Typically 57 to 64 abnormal and rearranged chromosomes
Neoplastic cells of canine transmissible venereal tumor display a highly characteristic chromosomal abnormality, containing typically fifty-seven to sixty-four abnormal and heavily rearranged chromosomes. This represents a significant reduction from the seventy-eight chromosomes found in normal canine somatic cells, proving that the tumor propagates as a clonal, transmissible allograft.

169. (D) High local invasiveness and recurrence with late or rare metastasis
Soft tissue sarcomas in veterinary medicine are grouped together because they exhibit similar clinical and biological behaviors. These mesenchymal tumors are characterized by high local invasiveness, a strong propensity for recurrence following incomplete surgical excision, and a relatively low or late rate of hematogenous metastasis, primarily spreading to the lungs.

170. (A) Antoni A and Antoni B tissue patterns
Peripheral nerve sheath tumors, such as Schwannomas, are characterized microscopically by distinct Antoni A and Antoni B tissue patterns. Antoni A areas consist of highly cellular, dense spindle cells arranged in palisades or whorls around eosinophilic zones, while Antoni B areas are hypocellular, loose, and myxoid in appearance with abundant extracellular matrix.

171. (B) Mesothelioma
A mesothelioma is a malignant neoplasm arising from the mesothelial cells lining the pleural, peritoneal, or pericardial cavities. In humans, this tumor is strongly linked to asbestos exposure, but it also occurs in animals, presenting as diffuse, multinodular growths along serosal surfaces, frequently causing significant pleural or peritoneal fluid accumulation.

172. (B) Gingiva of the oral cavity
Epulides are common, benign, exophytic neoplasms located strictly on the gingiva of the oral cavity, arising from the periodontal ligament or connective tissues of the jaw. These non-metastatic masses, such as peripheral odontogenic fibromas, can cause localized dental displacement and oral discomfort but are successfully managed with complete surgical excision.

NODIA

173. (A) Spindle cells in fingerprint whorls surrounding small vessels
A hemangiopericytoma is characterized histologically by neoplastic spindle cells arranged in distinctive, concentric, fingerprint-like whorls around small, thin-walled vascular channels.

diagnosing neoplasms. Unlike cytological methods, this technique preserves the overall tissue architecture, allowing pathologists to definitively identify the tumor type, evaluate the depth of invasion, assess vascular involvement, and verify if the surgical margins are completely free of neoplastic cells.

135. (B) Cytology lacks evaluation of tissue architecture and invasion
The primary limitation of cytology compared to histopathology is its inability to evaluate tissue architecture and local tissue invasion. Because cytological samples consist of isolated cells and small clusters, pathologists cannot determine if cells have breached the basement membrane, invaded local blood vessels, or disrupted the normal organization of the surrounding stroma.

136. (C) 10% Neutral Buffered Formalin
The standard, globally accepted fixative for routine surgical histopathology is ten percent neutral buffered formalin. This solution preserves tissue morphology by forming cross-links between amino acids, preventing autolysis and bacterial decomposition, while maintaining a neutral pH to protect cellular details and prepare the specimens for optimal paraffin embedding and sectioning.

137. (D) Hematoxylin and Eosin (H&E)
Hematoxylin and eosin is the universal staining method utilized for routine histopathological evaluation of tissue sections. Hematoxylin stains acidic cellular structures, such as nucleic acids in the nucleus, a deep blue-purple color, while eosin counterstains basic cytoplasmic proteins and extracellular collagen fibers varying shades of pink, highlighting tissue architecture.

138. (A) Chromogen-tagged antibodies binding to specific cellular antigens
Immunohistochemistry is a diagnostic technique that identifies specific cell lineages and proteins by utilizing chromogen-labeled antibodies. These engineered antibodies bind with high specificity to target antigens within a tissue section. The subsequent enzymatic reaction produces a colored precipitate, allowing pathologists to visualize the presence and localization of specific proteins microscopically.

139. (B) Epithelial (Carcinoma)
Cytokeratin is an intermediate filament protein expressed exclusively by epithelial cells. In cases of poorly differentiated or anaplastic tumors, detecting cytokeratin via immunohistochemistry provides definitive evidence of epithelial origin. This diagnostic finding distinguishes carcinomas from mesenchymal, melanocytic, or round cell neoplasms, which express different intermediate filament proteins.

140. (B) Mesenchymal
Vimentin is the primary intermediate filament protein found in cells of mesenchymal origin, such as fibroblasts, endothelial cells, and chondrocytes. In diagnostic pathology, immunohistochemical staining for vimentin is routinely utilized to identify tumors of mesenchymal lineage, effectively confirming a sarcoma diagnosis and ruling out carcinomas of epithelial origin.

141. (A) Specific genetic mutations, translocations, or viral DNA
The clinical application of polymerase chain reaction in oncology is highly valuable for identifying specific genetic abnormalities, gene translocations, or oncogenic viral DNA. This molecular technique amplifies trace amounts of target nucleic acids, enabling sensitive detection of diagnostic mutations, helping characterise tumor subtypes, and assisting in the selection of targeted therapies.

142. (C) Fluorescence In Situ Hybridization (FISH)
Fluorescence in situ hybridization is a powerful molecular cytogenetic technique used to detect and localize specific DNA sequences within intact tissue sections. By using fluorescently labeled probes that hybridize to target genes, this method allows pathologists to visualize chromosomal aberrations, gene amplifications, or translocations directly under a fluorescence microscope.

143. (D) Hematopoietic neoplasms like leukemia and lymphoma
Flow cytometry is a sophisticated laboratory technique routinely used for the immunophenotyping and subtyping of hematopoietic neoplasms, such as leukemia and lymphoma. By analyzing light scatter and fluorescence emission from single-cell suspensions labeled with fluorochrome-conjugated antibodies, it rapidly quantifies cellular size,

Click Here to Buy book on Amazon

due to cell loss or shrinkage, and from agenesis, which represents complete congenital absence.

184. (C) Both hypertrophy and hyperplasia
The physiological enlargement of the uterus during pregnancy is a classic example of both cellular hypertrophy and hyperplasia occurring simultaneously. Estrogenic stimulation causes uterine smooth muscle cells to increase in size by synthesizing more contractile proteins, while concurrently stimulating mitotic division to increase the total number of myometrial cells.

185. (D) Squamous metaplasia
Squamous metaplasia is a reversible adaptation where one mature, differentiated cell type is replaced by another mature cell type better suited to withstand chronic irritation. In the respiratory tract of chronic smokers, the delicate ciliated columnar epithelium is replaced by stratified squamous epithelium, providing protection but losing mucociliary clearance functions.

186. (A) Disuse atrophy
Atrophy of skeletal muscle following the application of a cast is classified as disuse atrophy. The immobilization of the limb removes the normal mechanical work and functional demand on the muscle fibers. This reduction in stimulation accelerates protein degradation via the ubiquitin-proteasome pathway, causing the muscle cells to shrink.

187. (B) Dysplasia
Dysplasia is a reversible cellular adaptation characterized by a loss of individual cell structural uniformity and architectural orientation within an epithelium. It is characterized by cellular pleomorphism, nuclear hyperchromasia, and increased mitotic figures. Although it is a pre-neoplastic change, it does not inevitably progress to frank cancer.

188. (B) Condensed and fragmented chromatin with intact membranes
Apoptosis is characterized morphologically by nuclear chromatin condensation and fragmentation, cellular shrinkage, and the formation of apoptotic bodies with intact plasma membranes. Unlike necrosis, which involves cell swelling, rapid membrane rupture, and release of intracellular contents, apoptosis does not trigger a surrounding inflammatory response in the host tissue.

189. (A) Squamous cell carcinoma
A malignant tumor arising from the stratified squamous epithelium of the skin is termed a squamous cell carcinoma. This neoplasm is characterized by invasive clusters of atypical squamous epithelial cells that breach the basement membrane and infiltrate the underlying dermis, often displaying cellular keratinization and prominent intercellular bridges.

190. (C) A malignant epithelial tumor that has not breached the membrane
Carcinoma in situ is defined as a pre-invasive malignant epithelial neoplasm that exhibits all the cellular features of malignancy but remains strictly confined to the epithelial layer. Because the neoplastic cells have not breached the basement membrane, there is no direct access to blood vessels or lymphatic channels.

191. (D) Benign
A benign neoplasm is clinically characterized by a slow growth rate, expansion rather than infiltration of surrounding tissues, and a well-defined, localized mass that is frequently encapsulated. These tumors do not metastasize to distant organs, and complete surgical excision typically results in a curative outcome for the patient.

192. (C) Proto-oncogene
Proto-oncogenes are normal, highly conserved cellular genes that play essential roles in regulating cell growth, division, and differentiation. When these genes undergo mutations, chromosomal translocations, or amplification, they can be converted into active oncogenes, which drive the unregulated, autonomous proliferation that is characteristic of malignant neoplastic cells.

193. (B) Inhibit the E_2F factor to block cell cycle progression
The normal retinoblastoma gene product acts as a critical tumor suppressor by regulating the transition from the G1 to the S phase of the cell cycle. In its active, hypophosphorylated state, the retinoblastoma protein binds to and inhibits E_2F transcription factors, preventing the transcription of genes required for DNA replication.

NODIA

infiltration, early hematogenous metastasis, and a strong propensity for acute, life-threatening rupture.

154. (A) Acute tumor rupture causing massive hemoabdomen
A classic, life-threatening clinical presentation of splenic hemangiosarcoma is acute tumor rupture, leading to massive and rapid blood loss into the peritoneal cavity, known as hemoabdomen. This catastrophic event results in acute hypovolemic shock, collapse, and pale mucous membranes, presenting as a surgical emergency in affected canine patients.

155. (B) Adipocytes
Lipomas are very common, benign mesenchymal neoplasms composed entirely of mature adipocytes. Microscopically, these tumors consist of well-differentiated fat cells that are indistinguishable from normal subcutaneous adipose tissue, organized into lobules by thin fibrous connective tissue septa, and typically display a slow, non-invasive growth pattern.

156. (B) Lymph nodes, spleen, and mucosa-associated lymphoid tissue
Lymphoma is a specialized malignant neoplasm involving the clonal proliferation of lymphoid cells that primarily arises within solid lymphoid organs. This includes the lymph nodes, spleen, thymus, and mucosa-associated lymphoid tissue. This presentation distinguishes lymphoma from leukemia, which primarily originates in the bone marrow and circulates within the blood.

157. (A) Histamine, heparin, and proteolytic enzymes
Mast cell tumors contain characteristic cytoplasmic granules filled with bioactive mediators, including histamine, heparin, and diverse proteolytic enzymes such as tryptase and chymase. The systemic release of these granules during tumor manipulation can trigger severe local inflammation, gastric ulceration, and systemic anaphylaxis, significantly influencing patient prognosis and clinical management.

158. (C) Toluidine Blue or Giemsa
Toluidine blue and Giemsa are specialized histological stains used to highlight the metachromatic cytoplasmic granules of mast cells. These basic dyes bind to the highly acidic heparin glycosaminoglycans within the granules, shifting the color from blue to purple-red, which facilitates clear differentiation of mast cell tumors from other round cell neoplasms.

159. (D) Oral cavity and mucocutaneous junctions
Melanomas in dogs exhibit highly variable biological behaviors depending on their anatomical location. Neoplasms arising from the oral cavity and mucocutaneous junctions are associated with the highest rates of malignancy, showing aggressive local infiltration, a high mitotic index, and a strong tendency for early metastasis to regional lymph nodes and lungs.

160. (C) An allograft transmitted by physical transfer of living tumor cells
Canine transmissible venereal tumor is a unique neoplasm characterized as a cellular allograft. It is transmitted directly between dogs during coitus or social contact through the physical transplantation of viable tumor cells onto mucous membranes. The tumor cells act as infectious agents, evading the host immune system to establish localized growths.

161. (B) Spontaneous regression driven by infiltrating host T cells
Canine cutaneous histiocytomas are benign epidermal dendritic cell tumors that occur in young dogs and are renowned for their spontaneous regression. This regression is driven by a robust host cell-mediated immune response, where infiltrating cytotoxic T lymphocytes target and destroy the neoplastic histiocytes, leading to complete resolution of the tumor.

162. (D) Bence Jones proteins
Multiple myeloma is a systemic, malignant proliferation of plasma cells that produce excessive monoclonal immunoglobulins or light chains. These free immunoglobulin light chains, known as Bence Jones proteins, are small enough to pass through the glomerular filtration barrier and accumulate in the urine, serving as an important diagnostic hallmark of this disease.

163. (A) Adenomas exhibit organized architecture lacking stromal/vascular invasion
The primary microscopic distinction between a benign adenoma and a malignant adenocarcinoma is the structural organization and absence of tissue invasion. Adenomas maintain a well-organized glandular architecture with an intact basement membrane and display no stromal or vascular invasion, whereas adenocarcinomas

Downloaded from www.pdf.tube

pulmonary neoplasms, metastatic tumors, or chronic inflammatory masses, mediated via vagal nerve stimulation and altered blood flow.

125. (A) Coagulation system (migratory thrombophlebitis)
Trousseau's syndrome is a paraneoplastic disorder involving the coagulation system, characterized by recurrent, migratory thrombophlebitis and thromboembolism. This hypercoagulable state is induced by the tumor-specific release of procoagulants, tissue factor, and inflammatory cytokines, which interact with host platelets and endothelial cells to promote spontaneous clotting in blood vessels.

126. (C) Gastric ulceration
Functional mast cell tumors release large quantities of histamine into systemic circulation, leading to severe gastrointestinal complications. Histamine binds to histamine-2 receptors on gastric parietal cells, stimulatng excessive hydrochloric acid secretion. This chronic hyperacidity damages the gastric mucosa, frequently resulting in severe, life-threatening gastroduodenal ulceration and gastrointestinal bleeding.

127. (D) Cellular differentiation and mitotic activity of the tumor cells
Tumor grading is a histopathological assessment that evaluates the biological aggressiveness of a neoplasm based on microscopic characteristics of the cells. Pathologists determine the grade by analyzing cellular differentiation, pleomorphism, nuclear atypia, and the mitotic index, which helps predict the clinical behavior, growth rate, and overall metastatic potential of the tumor.

128. (C) Physical size and local invasiveness of the primary tumor
In the TNM staging system, the "T" component represents the primary tumor. This parameter evaluates the physical size, volume, and depth of local tissue invasion of the initial neoplastic mass. Accurately assessing this factor provides essential information regarding the local extent of the disease, guiding surgical planning and prognostic determinations.

129. (B) No regional lymph node metastasis
The designation "N0" in the TNM clinical staging system indicates that there is no evidence of tumor metastasis to the regional lymph nodes. This finding suggests the neoplasm remains localized to the primary site, representing a lower stage of disease progression compared to stages where nodal involvement is confirmed.

130. (D) A large tumor with regional nodal involvement and metastasis
A tumor staged as T3, N2, M1 represents an advanced stage of malignant neoplastic disease. The designation indicates a large or deeply invasive primary tumor, significant involvement of regional lymph nodes, and the confirmed presence of distant hematogenous metastasis, collectively indicating a poor prognosis and requiring aggressive systemic therapy.

131. (A) Grading evaluates cellular behavior; staging measures anatomical disease extent
Tumor grading and clinical staging assess different aspects of neoplastic disease to guide prognosis. Grading is a microscopic evaluation of cellular differentiation and proliferative activity performed by a pathologist. Conversely, staging is a clinical determination that measures the anatomical extent and physical spread of the tumor throughout the patient's body.

132. (C) Spontaneously shed from epithelial surfaces or into body fluids
Exfoliative cytology is a diagnostic method based on the microscopic examination of cells that have spontaneously detached or shed from epithelial surfaces and mucous membranes. These free cells are typically collected from body fluids, secretions, or brushings, allowing for a minimally invasive assessment of inflammatory, hyperplastic, or neoplastic changes.

NODIA

133. (A) Individual cell morphology of a palpable mass via a thin needle
Fine needle aspiration cytology is a rapid, minimally invasive diagnostic technique used to evaluate palpable tissue masses. By utilizing a thin-gauge needle to aspirate individual cells and small cellular aggregates, this procedure allows clinicians to evaluate cellular morphology, distinguish inflammatory from neoplastic processes, and often identify specific tumor lineages.

134. (D) Surgical histopathology of an excisional tissue biopsy
Histopathological examination of an excisional tissue biopsy remains the gold standard for

This unique microscopic architecture, arising from vascular pericytes, is a key diagnostic feature utilized by pathologists to differentiate this tumor from other soft tissue sarcomas of the skin and subcutis.

174. (C) Bile ducts
Cholangiocarcinomas are highly malignant epithelial neoplasms that originate from the epithelial cells of the intrahepatic or extrahepatic bile ducts. These tumors are characterized by an abundant desmoplastic stroma and a glandular growth pattern, frequently leading to localized bile duct obstruction, cholestasis, liver dysfunction, and early metastasis to regional lymph nodes.

175. (D) Coagulative necrosis
Coagulative necrosis is the primary type of tissue death observed within rapidly growing solid tumors that outgrow their blood supply. When neoplastic expansion surpasses the rate of angiogenesis, localized ischemia occurs. This leads to cell death while temporarily preserving the basic structural outlines of the dead cells and tissue architecture.

176. (C) Scirrhous carcinomas
Desmoplasia is a prominent, reactive fibrotic stroma characterized by intense collagen deposition and activated myofibroblasts. This response is most commonly induced by scirrhous carcinomas, such as certain mammary or gastrointestinal adenocarcinomas, where the invasive epithelial tumor cells secrete growth factors that stimulate host fibroblasts to produce a dense, firm extracellular matrix.

177. (B) Congo Red
Confirming the presence of amyloid associated with multiple myeloma relies on Congo Red staining. When viewed under a polarized light microscope, amyloid fibrils stained with Congo Red display a pathognomonic apple-green birefringence. This optical property is due to the highly ordered, beta-pleated sheet configuration of the misfolded protein aggregates.

178. (D) Actively dividing with diminished DNA repair capacity
Ionizing radiation selectively targets and destroys neoplastic cells that are actively dividing and possess diminished DNA repair capabilities. The radiation induces lethal double-stranded DNA breaks and generates reactive oxygen species. Because tumor cells often lack robust DNA repair mechanisms, they undergo mitotic catastrophe or apoptosis when attempting cell division.

179. (A) Telomerase
Malignant tumors bypass the Hayflick limit of cellular senescence by reactivating the enzyme telomerase. This specialized DNA polymerase maintains telomere length at the ends of chromosomes during successive cell divisions, preventing chromosomal instability and cellular aging, thereby conferring unlimited replicative potential and replicative immortality to the neoplastic cell population.

180. (C) Apocrine gland anal sac adenocarcinoma
Hypercalcemia of malignancy in dogs is most classically associated with apocrine gland anal sac adenocarcinoma. This highly invasive epithelial tumor frequently secretes parathyroid hormone-related protein, which mimics parathyroid hormone. This ectopic secretion causes profound bone resorption and renal calcium retention, resulting in life-threatening elevations in systemic blood calcium concentrations.

181. (A) Thymoma
Myasthenia gravis is a classic neuromuscular paraneoplastic syndrome most commonly associated with thymomas. This autoimmune disorder occurs when the neoplasm triggers the production of autoantibodies that target and destroy acetylcholine receptors at the neuromuscular junction, leading to progressive muscle weakness, exercise intolerance, and often secondary megaesophagus.

182. (D) Hypertrophy
Hypertrophy is a cellular adaptation characterized by an increase in the physical size of individual cells, resulting in the enlargement of the organ. This process occurs without cell division and is driven by an increased synthesis of structural proteins and organelles, typically in response to increased workload or hormonal stimulation.

183. (B) Never reached normal mature size during development
Hypoplasia is a developmental defect characterized by an organ or tissue that fails to reach its normal mature size during growth. This differs from atrophy, where a fully developed, mature organ subsequently decreases in size

Click Here to Buy book on Flipkart

secondary tumors, making the third statement incorrect.

106. (A) Leukocyte adhesion and migration
Tumor cell extravasation at distant metastatic sites mimics the physiological process of leukocyte adhesion and migration during inflammation. Neoplastic cells in the circulation attach to the vascular endothelium via adhesion molecules, such as selectins and integrins, before transmigrating across the endothelial barrier into the surrounding parenchyma to establish secondary colonies.

107. (B) Metastasis success depends on tumor cell properties interacting with favorable target organ microenvironments
The seed and soil hypothesis asserts that successful metastasis requires compatible interactions between circulating tumor cells and the destination organ microenvironment. Tumor cells act as seeds that can only survive, colonize, and proliferate in specific host tissues, which serve as receptive soil by providing the necessary growth factors, extracellular matrix components, and cellular support.

108. (B) Cells survive in a non-proliferative state as solitary cells or micrometastases
Dormancy describes a clinical stage where disseminated tumor cells remain viable at secondary sites but do not actively proliferate or form macroscopic lesions. These cells can persist as quiescent single cells or tiny, clinically undetectable micrometastases, balanced by equal rates of cell division and apoptosis, until environmental changes trigger reactivation.

109. (A) Expressed only by tumor cells and not by any normal host cells
Tumor-specific antigens are unique molecules expressed exclusively on the surface of neoplastic cells and are entirely absent from normal host tissues. These proteins typically arise from somatic mutations, abnormal splicing, or oncogenic viral proteins, making them highly specific targets for recognition and destruction by the host adaptive immune system.

110. (C) I only
Tumor-associated antigens are normal self-proteins that are expressed inappropriately or at abnormally high levels by neoplastic cells. Because they are native host proteins, they are not entirely foreign to the immune system. Examples include fetal proteins expressed in adulthood, such as alpha-fetoprotein, and normal proteins that undergo overexpression during oncogenesis.

111. (D) Colon
Carcinoembryonic antigen is a highly characterized oncofetal glycoprotein primarily utilized as a clinical biomarker for adenocarcinomas of the colon. Although normally expressed during fetal gut development, its production is heavily upregulated in malignant colorectal tissues, making it useful for monitoring tumor progression, recurrence, and response to therapeutic interventions.

112. (C) Tumor-specific antigens (TSAs)
Antigens encoded by oncogenic viruses, such as the E6 and E7 proteins of human papillomavirus, are classified as tumor-specific antigens. Since these viral proteins are completely absent from healthy, uninfected host cells, they serve as unique foreign markers that can trigger a robust, targeted cytotoxic T lymphocyte response against the infected neoplastic cells.

NODIA

113. (B) $CD8^+$ Cytotoxic T lymphocytes (CTLs)
Cytotoxic T lymphocytes expressing CD8 are the primary immune cells responsible for recognizing and directly eliminating neoplastic cells. These lymphocytes utilize T-cell receptors to detect foreign or abnormal peptide antigens displayed on major histocompatibility complex class I molecules, subsequently inducing target cell apoptosis through the release of perforin and granzymes.

114. (D) Downregulated or lost surface MHC Class I expression
Natural killer cells serve as critical innate immune monitors by identifying and destroying abnormal cells that have lost or downregulated their surface major histocompatibility complex class I molecules. This mechanism, known as missing-self recognition, prevents tumor cells from escaping adaptive cytotoxic T-cell detection through the selective downregulation of antigen-presenting machinery.

115. (A) I, II, IV and V
Neoplastic cells employ diverse strategies to escape immune destruction, including the downregulation of major histocompatibility

194. (D) Cancer develops from multiple genetic mutations over time
The multi-hit hypothesis of carcinogenesis establishes that the development of cancer is a multi-step process requiring the accumulation of multiple independent genetic mutations over time. A single genetic alteration is rarely sufficient to induce malignancy; instead, sequential mutations must occur in proto-oncogenes, tumor suppressor genes, and DNA repair pathways.

195. (A) Slow-transforming retroviruses
Slow-transforming retroviruses cause neoplastic transformation through insertional mutagenesis, a process requiring the integration of viral DNA near a host proto-oncogene. This integration alters host gene expression, driving clonal expansion. Because this event occurs randomly and requires subsequent secondary mutations, these viruses are characterized by a long clinical latent period.

196. (B) Induces clonal expansion and proliferation of a previously initiated cell
In chemical carcinogenesis, a promoter is a non-mutagenic agent that stimulates the clonal expansion and proliferation of a cell previously mutated by an initiator. While promoters do not directly damage DNA, they create a proliferative environment that increases the likelihood of acquiring additional mutations necessary for full neoplastic transformation.

197. (B) PCR for Antigen Receptor Rearrangement (PARR)
PCR for Antigen Receptor Rearrangement is a molecular diagnostic technique used to distinguish neoplastic from reactive lymphoid proliferations. By amplifying the variable regions of immunoglobulin or T-cell receptor genes, it determines if a lymphocyte population is monoclonal, indicating a lymphoma, or polyclonal, which is characteristic of reactive hyperplasia.

198. (D) Depth of invasion, mitoses, and cell morphology
Histopathological grading of mast cell tumors evaluates specific microscopic criteria to predict clinical outcome. Pathologists assess parameters such as the mitotic count per high-power fields, cellular and nuclear pleomorphism, karyomegaly, multinucleation, and the depth of tumor invasion into adjacent subcutaneous tissues, which correlate with biological behavior and metastatic potential.

199. (B) Variability in size and shape
A diagnostic finding of pleomorphism in a cytology report indicates significant variability in the size and shape of cells and their nuclei. This variation, representing anisocytosis and anisokaryosis, is a classic cytological hallmark of malignancy, reflecting the loss of normal genetic and cellular regulatory mechanisms.

200. (A) Vascular Endothelial Growth Factor (VEGF)
Vascular endothelial growth factor is a potent pro-angiogenic cytokine secreted by hypoxic tumor cells in response to hypoxia-inducible factor-one. This protein binds to specific tyrosine kinase receptors on nearby endothelial cells, stimulating their proliferation, migration, and survival, which drives the neoangiogenesis necessary to sustain tumor growth.

201. (D) Metalloproteinases (MMPs)
Neoplastic cells secrete matrix metalloproteinases, a group of zinc-dependent endopeptidases, to degrade the basement membrane and surrounding extracellular matrix. By cleaving structural proteins like collagen and laminin, these enzymes break down physical barriers, facilitating tumor cell invasion into adjacent tissues and promoting entry into blood and lymphatic vessels.

202. (A) The lung provides the first major capillary bed for venous drainage
The lung is the most common site for hematogenous metastasis because it provides the first major capillary bed encountered by venous blood draining from most organs. Circulating tumor cells that enter the systemic veins lodge mechanically within the small, narrow pulmonary capillaries, where they can extravate and colonize the lung tissue.

203. (B) Transcoelomic seeding
Transcoelomic seeding is a major route of metastasis characterized by the shedding of neoplastic cells into anatomical body cavities. Ovarian carcinomas frequently spread via this mechanism, releasing cells into the peritoneal fluid. These cells then implant and proliferate across the abdominal and peritoneal serosal surfaces without necessarily invading deeply into organs.

NODIA

2007

45. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): A syndromic diagnosis groups concurrent clinical and pathological findings into a recognized physiological disorder or syndrome.
Reason (R): It identifies the exact causative pathogen directly from the macroscopic lesions without needing laboratory confirmation.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2012

46. Under the Prevention and Control of Infectious and Contagious Diseases in Animals (PCICDA) Act, 2009, what is a mandatory obligation of a veterinarian upon encountering a scheduled notifiable disease outbreak?
(A) To immediately vaccinate all of the susceptible animals in the entire district
(B) To independently declare an official international trade embargo on livestock
(C) To report the outbreak to the designated local or state authority
(D) To cull all potentially infected animals prior to official confirmation

2003

47. Which of the following is NOT a typical objective of the PCICDA Act, 2009?
(A) Prevention of the spread of infectious animal diseases
(B) Control and eradication of scheduled diseases
(C) Declaration of controlled and eradicated areas
(D) Regulation of veterinary clinic registration fees

2010

48. In the context of veterinary epidemiology and the PCICDA Act, which term defines the constant presence of a disease within a given geographic area?
(A) Epidemic
(B) Pandemic
(C) Endemic
(D) Sporadic

2003

49. A key advantage of compulsory notification laws for animal diseases is that they:
(A) Eliminate any immediate clinical need for routine or advanced diagnostic testing
(B) Allow government mobilization of resources for containment and stamping out
(C) Prevent field veterinarians from handling potentially infectious cases entirely
(D) Automatically provide financial compensation to farmers without official verification

2019

50. Match the types of diagnoses in List I with their corresponding examples in List II.
List I: (Diagnostic Type)
P. Morphological diagnosis
Q. Etiological diagnosis
R. Syndromic diagnosis
S. Clinical diagnosis
List II: (Example)
1. Right-sided congestive heart failure
2. Parvoviral enteritis
3. Severe, acute, diffuse, purulent bronchopneumonia
4. Suspected canine distemper based on ocular discharge and seizures
(A) P-1, Q-2, R-3, S-4
(B) P-3, Q-1, R-4, S-2
(C) P-3, Q-2, R-1, S-4
(D) P-2, Q-3, R-4, S-1

2025

51. What is the standard purpose of veterinary quarantine protocols?
(A) To isolate animals to observe for infectious diseases before integration
(B) To permanently segregate healthy native animals from newly introduced breeds
(C) To provide a highly controlled clinical environment exclusively for vaccination
(D) To house animals after clinical recovery until their meat is fit for consumption

2013

52. The duration parameter for veterinary quarantine is typically determined by the:
(A) Estimated natural physiological lifespan of the animal species
(B) Average expected daily weight gain of the quarantined animal
(C) Maximum incubation period of the disease of concern
(D) Total financial cost of feeding the animal in the facility

Downloaded from www.pdf.tube

CHAPTER 07

Postmortem Diagnosis and Histopathological Techniques

2017

1. Which of the following best defines somatic death in veterinary pathology?
(A) The localized death of cell populations within a living organism due to injury
(B) The complete and irreversible cessation of all vital functions of the body
(C) The progressive degradation of individual cells and tissues by autolytic enzymes
(D) The gradual depletion of cellular ATP leading to uncoordinated muscle contraction

2009

2. Following somatic death, the chronological sequence of early postmortem changes typically occurs in which order?
(A) Livor mortis, algor mortis, rigor mortis
(B) Algor mortis, rigor mortis, livor mortis
(C) Rigor mortis, livor mortis, algor mortis
(D) Algor mortis, livor mortis, rigor mortis

2024

3. What is the fundamental biochemical mechanism responsible for the onset of rigor mortis?
(A) The postmortem depletion of ATP preventing the dissociation of actin and myosin filaments
(B) The accumulation of lactic acid causing coagulation of sarcoplasmic proteins
(C) The enzymatic degradation of the Z-lines in muscle fibers by endogenous proteases
(D) The passive diffusion of calcium ions out of the sarcoplasmic reticulum triggering autolysis

2010

4. Which of the following is the correct statement regarding molecular death?
(A) It occurs simultaneously with somatic death across all tissues.
(B) It precedes somatic death and triggers systemic organ failure.
(C) It refers to the sequential death of individual cells and tissues following somatic death.
(D) It is characterized by the sudden cessation of the cardiovascular and respiratory systems.

2005

5. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): Livor mortis is typically less pronounced in animals that die of severe hemorrhage.
Reason (R): Livor mortis depends on the gravitational settling of blood in dependent capillaries, which requires an adequate blood volume.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

NODIA

2016

6. Which of the following is a primary characteristic of putrefaction rather than autolysis?
(A) It is primarily driven by the action of active endogenous cellular hydrolytic enzymes.
(B) It occurs symmetrically and uniformly across all sterile parenchymal organs in the body.
(C) It requires a highly oxygen-rich environment to proceed optimally within closed cavities.
(D) It is characterized by tissue degradation by saprophytic bacteria and their enzymes.

2004

7. Which postmortem artifact is caused by the interaction of hydrogen sulfide gas produced by bacteria with iron from degraded hemoglobin?
(A) Postmortem imbibition
(B) Pseudomelanosis
(C) Hypostatic congestion
(D) Chicken fat clot

2005

8. Which of the following is an incorrect statement regarding the differentiation between antemortem thrombi and postmortem blood clots?

I. The proventriculus represents the true glandular stomach of the bird.
II. The bursa of Fabricius is located dorsal to the cloaca.
III. Birds possess a well-developed diaphragm that must be punctured to check for negative thoracic pressure.
IV. The crop is an expansion of the esophagus used for food storage.
V. Avian lungs are highly expansile and collapse completely upon opening the thorax.

Which of the above statements is/are correct?
(A) I, II and IV
(B) I, III, IV and V
(C) II, III and V
(D) I, II, III and IV

2022
26. Which anatomical sequence accurately reflects the direction of ingesta flow in the avian digestive tract, important for systemic examination?
(A) Crop → Ventriculus → Proventriculus → Small intestine
(B) Crop → Proventriculus → Ventriculus → Small intestine
(C) Proventriculus → Crop → Ventriculus → Small intestine
(D) Ventriculus → Crop → Proventriculus → Small intestine

2001
27. The objective documentation of gross lesions in descriptive pathology requires describing the lesion's size, shape, color, consistency, and:
(A) Microscopic architecture
(B) Distribution
(C) Genetic mutation profile
(D) Vector competence

2002
28. A lesion described as "friable" provides objective information regarding which descriptive parameter?
(A) Color
(B) Consistency
(C) Shape
(D) Size

2019
29. If a pathology report describes a lesion as "multifocal to coalescing," it is primarily detailing the lesion's:
(A) Distribution
(B) Volume
(C) Consistency
(D) Topography

2013
30. Which of the following is the correct sequence of descriptive terms applied when evaluating a pathological organ systematically?
(A) Microscopic features, organ weight, DNA analysis
(B) Organ identity, size/weight, color, consistency, distribution of lesions
(C) Etiological diagnosis, clinical history, syndromic classification
(D) Fixation time, embedding medium, microtome clearance angle

2025
31. Which descriptive term best fits a gross lesion that is elevated, solid, and larger than a papule?
(A) Macule
(B) Vesicle
(C) Ulcer
(D) Nodule

2017
32. In descriptive pathology, a lesion presenting as a central depression resembling a navel is termed:
(A) Pedunculated
(B) Sessile
(C) Umbilicated
(D) Verrucous

2008
33. Which gross lesion documentation practice is generally considered inappropriate for a formal necropsy report?
(A) Providing three-dimensional measurements in centimeters
(B) Using household items like "fruit-sized" to describe lesion size
(C) Describing the percentage of the organ affected by the lesion
(D) Noting the presence of distinct demarcations or borders

2015
34. What does the term "pathognomonic lesion" signify in veterinary pathology?
(A) A lesion that is highly non-specific and commonly found across multiple disease entities
(B) A lesion that only appears in the early, subclinical developmental stages of a disease
(C) A postmortem artifact that closely mimics severe, antemortem physical trauma
(D) A lesion characteristic of a single disease that allows immediate diagnosis

Click Here to Buy book on Amazon

2009

16. What is the primary advantage of performing a cosmetic necropsy on a companion animal?
(A) It significantly reduces the time required for extracting thoracic organs.
(B) It completely eliminates the potential risk of zoonotic disease transmission.
(C) It preserves the external carcass appearance for owner viewing or burial.
(D) It requires no highly specialized surgical tools or complex systemic incisions.

2017

17. The ideal recumbency for conducting a routine necropsy on a canine or feline patient is:
(A) Left lateral recumbency
(B) Dorsal recumbency
(C) Right lateral recumbency
(D) Ventral recumbency

2012

18. When performing a necropsy on a laboratory mouse, what specific technique is commonly employed prior to opening the body cavities to ensure optimal tissue preservation for histopathology?
(A) Submerging the intact carcass in 10% neutral buffered formalin for 48 hours
(B) Freezing the carcass at -80°C for 24 hours
(C) Injecting concentrated sulfuric acid into the abdominal cavity
(D) Performing whole-body perfusion with a fixative via the left ventricle

2019

19. What is a critical anatomical consideration when examining the gastrointestinal tract of swine during necropsy?
(A) The spiral colon located in the left abdominal cavity
(B) The physiological presence of a highly developed rumen
(C) The anatomical lack of a distinct gallbladder structure
(D) The total absence of a cardiac glandular stomach region

2007

20. Consider the following statements regarding species-specific necropsy techniques:
I. Swine are typically positioned in dorsal recumbency.
II. The brain of a carnivore is extracted by making a single transverse cut across the frontal bone.
III. In laboratory rodents, the thoracic cavity is opened before the abdominal cavity to maintain negative pressure.
IV. Carnivore plucks are removed starting from the mandibular symphysis down to the diaphragm.
Which of the above statements is/are incorrect?
(A) I and IV
(B) II and III
(C) II, III and IV
(D) I, II and IV

2015

21. Which organ is characteristically absent and therefore not evaluated during a routine equine necropsy?
(A) Gallbladder
(B) Spleen
(C) Pancreas
(D) Cecum

NODIA

2010

22. In avian necropsy, what is the primary purpose of wetting the carcass with water containing detergent prior to making incisions?
(A) To rapidly cool the carcass core and halt progressive postmortem autolysis
(B) To soften the thick epidermal skin layers for smoother scalpel penetration
(C) To facilitate the gross visualization of underlying subcutaneous fat deposits
(D) To prevent aerosolization of feathers and infectious pathogens

2015

23. During poultry necropsy, what specific anatomical structure is evaluated by reflecting the skin over the craniodorsal aspect of the tail base?
(A) Bursa of Fabricius
(B) Uropygial gland
(C) Proventriculus
(D) Harderian gland

2014

24. Which condition is most strongly suspected if significant asymmetrical enlargement of the sciatic nerve is observed during a poultry necropsy?
(A) Avian Influenza
(B) Newcastle Disease
(C) Marek's Disease
(D) Infectious Bursal Disease

2005

25. Consider the following statements concerning avian necropsy:

(A) Antemortem thrombi are typically firmly attached to the endothelial lining of blood vessels.
(B) Postmortem clots are characteristically friable, dull, and unattached to any vessel walls.
(C) Postmortem clots are composed entirely of leukocytes with no erythrocyte involvement.
(D) Antemortem thrombi possess a dull, granular surface and are quite firm in their consistency.

2005

9. Hemoglobin imbibition is most easily differentiated from antemortem hemorrhage by which of the following features?
(A) Hemoglobin imbibition is characterized by showing focal, sharply demarcated lesion borders.
(B) Antemortem hemorrhage is notable because it lacks true intact erythrocytes on microscopy.
(C) Antemortem hemorrhage only occurs in the endocardium and the tunica intima of large vessels.
(D) Hemoglobin imbibition diffuses into tissues without vascular rupture or inflammation.

2022

10. Match the postmortem artifacts in List I with their primary mechanisms in List II.

List I: (Artifact)
P. Algor mortis
Q. Pseudomelanosis
R. Livor mortis
S. Rigor mortis

List II: (Mechanism)
1. Settling of blood in dependent areas
2. Equilibration of body temperature with the environment
3. Depletion of cellular ATP
4. Reaction of bacterial hydrogen sulfide with iron

(A) P-2, Q-3, R-1, S-4
(B) P-2, Q-4, R-1, S-3
(C) P-1, Q-4, R-2, S-3
(D) P-3, Q-1, R-4, S-2

2002

11. Which standard anatomical position is predominantly used for the systematic necropsy of a large adult ruminant?
(A) Right lateral recumbency
(B) Left lateral recumbency
(C) Dorsal recumbency
(D) Ventral recumbency

2004

12. During a systemic equine necropsy, the animal is typically placed in left lateral recumbency. What is the primary anatomical advantage of this orientation?
(A) To facilitate rapid and immediate access to the left lung lobes and the left heart atrium
(B) To allow the large colon and cecum (situated primarily on the right) to be easily accessed and exteriorized
(C) To allow unobstructed clinical access to the descending colon and the terminal rectum
(D) To ensure that the liver can be extracted before removing other gastrointestinal organs

2011

13. Which procedure is least relevant during the initial external examination of a large ruminant carcass?
(A) Decalcifying the entire skull
(B) Evaluating the animal's overall body condition score
(C) Examining all natural external orifices for discharges
(D) Assessing the structural integrity of the hoof wall

2015

14. In ruminant necropsy, removing the "pluck" refers to the en bloc extraction of which organs?
(A) Liver, spleen, stomach, and intestines
(B) Kidneys, ureters, bladder, and urethra
(C) Tongue, larynx, trachea, lungs, and heart
(D) Brain, spinal cord, and meninges

2007

15. Consider the following statements regarding large animal necropsy procedures:
I. The rumen in cattle should be opened last to prevent contamination of the abdominal cavity.
II. In equines, the gastrointestinal tract is entirely removed as a single continuous unit without ligations.
III. The sciatic nerve is routinely examined by incising the medial aspect of the thigh.

Which of the above statements is/are correct?
(A) I and II
(B) I and III
(C) I only
(D) II and III

Downloaded from www.pdf.tube

2002
35. Arrange the following diagnostic steps in the logical sequence of postmortem evaluation:
I. Formulating an etiological diagnosis
II. Detailed description of gross lesions
III. Identifying morphological changes
IV. Synthesizing a syndromic diagnosis
(A) II, III, IV, I
(B) I, II, III, IV
(C) III, II, I, IV
(D) IV, III, II, I

2019
36. The presence of "button ulcers" in the large intestine of swine is considered a highly characteristic macroscopic lesion of which disease?
(A) Swine Erysipelas
(B) Porcine Circovirus Type 2
(C) Classical Swine Fever
(D) African Swine Fever

2010
37. Which of the following is a classic pathognomonic lesion associated with acute Rinderpest in cattle?
(A) "Zebra striping" of the large intestinal mucosa
(B) "Turkey egg" appearance of the kidneys
(C) "Nutmeg" appearance of the liver
(D) Wooden tongue

2017
38. A "nutmeg liver" is a gross pathological descriptor indicating chronic passive congestion. This macroscopic lesion is predominantly associated with failure of which system?
(A) Left side of the heart
(B) Renal glomeruli
(C) Pulmonary alveolar macrophages
(D) Right side of the heart

2005
39. In a necropsy, finding vesicles and subsequent erosions on the tongue, dental pad, and coronary bands of a cow is pathognomonic for:
(A) Blackquarter
(B) Foot-and-Mouth Disease
(C) Actinobacillosis
(D) Bovine Viral Diarrhea

2002
40. Consider the following statements regarding pathognomonic lesions and their associated diseases:
I. "Turkey egg" kidneys are characteristic of Classical Swine Fever.
II. "Wooden tongue" is caused by Actinobacillus lignieresii.
III. Diffuse severe splenic enlargement with dark, unclotted blood is classic for Anthrax.
IV. "Diamond skin" lesions in swine are pathognomonic for Pseudorabies.
Which of the above statements is/are incorrect?
(A) I and II
(B) II and III
(C) I, II and IV
(D) IV only

2008
41. Severe, acute, fibrinous pleuropneumonia with prominent interlobular edema (marbling) in a bovine lung is the classic macroscopic presentation of:
(A) Bovine Tuberculosis
(B) Infectious Bovine Rhinotracheitis
(C) Johne's Disease
(D) Contagious Bovine Pleuropneumonia (CBPP)

2014
42. A well-structured necropsy report should transition smoothly from descriptive findings to a diagnostic conclusion. Which of the following components forms the core of a morphological diagnosis?
(A) The organ affected, pathological process, distribution, severity, and duration
(B) The specific taxonomical classification of the infectious agent causing the disease
(C) A highly detailed clinical history provided in writing by the primary veterinarian
(D) A comprehensive summary of laboratory diagnostic test results confirming the toxin

2013
43. Formulating an etiological diagnosis differs from a morphological diagnosis in that the etiological diagnosis explicitly names the:
(A) Severity and chronicity of the lesion
(B) Causative agent responsible for the lesions
(C) Anatomic location of the tissue damage
(D) Functional failure leading to death

NODIA

2005
44. Based on the descriptive phrase "severe, acute, diffuse, fibrinohemorrhagic enteritis", what type of diagnosis is this?
(A) Etiological diagnosis
(B) Morphological diagnosis
(C) Clinical diagnosis
(D) Syndromic diagnosis

2011

61. If an animal is suspected of dying from malicious strychnine poisoning, which gross clinical finding is most expected prior to death?
(A) Tetanic convulsions and extreme extensor rigidity
(B) Profuse hemorrhagic diarrhea
(C) Severe icterus and hepatic necrosis
(D) Symmetrical alopecia and skin thickening

2004

62. Which organ is considered the most crucial for collection during a suspected malicious poisoning case due to its role in biotransformation and accumulation of toxins?
(A) Spleen
(B) Lungs
(C) Liver
(D) Heart

2003

63. Under the triple packaging system for dispatching biological specimens, what is the primary function of the intermediate (secondary) packaging?
(A) To provide the main rigid defense mechanism against external physical crushing forces
(B) To secure and hold the clinical history form directly in contact with the tissue specimen
(C) To serve as a high-capacity thermal barrier using pressurized liquid nitrogen
(D) To contain leaks from the primary receptacle and hold absorbent material

2022

64. What standard code is mandated for marking the outer packaging of Category B infectious substances shipped internationally?
(A) UN 2814
(B) UN 1203
(C) UN 3373
(D) UN 1075

2008

65. Consider the following statements regarding specimen dispatch:
I. Tissues for molecular analysis (PCR) should be preserved in 10% formalin.
II. The quantity of absorbent material must be sufficient to absorb the entire fluid content of the primary receptacle.
III. Tissues for microbiological culture should be dispatched strictly in a sterile container on ice.
IV. Blood smears for hemoprotozoan diagnosis should be fixed in methanol before dispatch.
V. Ice packs used for cooling should be placed directly inside the primary receptacle with the tissue.
Which of the above statements is/are incorrect?
(A) I and II
(B) I and V
(C) III and IV
(D) II, III and V

2022

66. To preserve biological specimens specifically for molecular analysis (e.g., RNA extraction), which stabilizing agent is most appropriate?
(A) 10% Neutral Buffered Formalin
(B) Bouin's fluid
(C) Saturated sodium chloride
(D) RNAlater

2024

67. The principle of tissue fixation involves which primary biochemical mechanism?
(A) The cross-linking of proteins to halt autolysis
(B) The active enzymatic degradation of structural cellular proteins
(C) The rapid removal of structural water from tissues using alcohol
(D) The selective decalcification of the heavy calcium bone matrix

2024

68. What is the optimal volume ratio of fixative to tissue required to ensure adequate and uniform fixation?
(A) 1:1
(B) 2:1
(C) 5:1
(D) 10:1 to 20:1

2018

69. Formalin pigment (acid hematin) is a common fixation artifact. Under what conditions is it most likely to form?
(A) When blood-rich tissues are fixed in unbuffered, acidic formalin
(B) When specimens are preserved primarily in highly alkaline solution mixtures
(C) When tissues are excessively over-dehydrated during xylene clearance steps
(D) When tissue paraffin blocks are sectioned too thickly on the microtome

2024

70. Which of the following is the correct sequence of steps involved in routine tissue processing for histopathology?
(A) Clearing, Dehydration, Impregnation,

Click Here to Buy book on Flipkart

2003

53. Which document formally attests that an animal has been examined by a qualified veterinarian and shows no clinical signs of infectious or contagious disease?
(A) Soundness and health certificate
(B) Certificate of origin
(C) Import permit
(D) Bill of lading

2013

54. If a suspected exotic disease is detected during the quarantine period, the most appropriate immediate action is to:
(A) Release the suspect animal immediately to build herd immunity in the area
(B) Administer high doses of broad-spectrum antibiotics and release the animal
(C) Relocate the clinical animal directly to a general veterinary hospital unit
(D) Halt all quarantine station movement and notify statutory authorities

2004

55. Consider the following statements regarding vetero-legal necropsies:
I. Maintaining the chain of custody for all samples is legally mandatory.
II. A police officer or magistrate must generally authorize a vetero-legal necropsy.
III. Photographs taken during the necropsy require no labeling or scaling to be admissible in court.
Which of the above statements is/are correct?
(A) I and III
(B) II and III
(C) I and II
(D) I only

2006

56. What is the fundamental requirement of establishing a "chain of custody" in vetero-legal cases?
(A) Using physical heavy metal chains to secure the animal carcass to the necropsy table
(B) Ensuring that the animal's owner is physically present during the entire laboratory analysis
(C) Documenting uninterrupted tracking of evidence from collection to disposition
(D) Sending the collected evidence to at least three independent laboratories simultaneously

2019

57. Which type of preservative is explicitly recommended for dispatching visceral organs for toxicological analysis in cases of suspected heavy metal poisoning?
(A) 10% Neutral Buffered Formalin
(B) 70% Ethyl alcohol
(C) Bouin's fluid
(D) Saturated solution of common salt (NaCl)

2024

58. In cases involving insurance claims, the vetero-legal necropsy must uniquely emphasize the documentation of:
(A) Specific identification marks, brands, tags, and microchips
(B) The exact quantitative load of various parasitic worms in the gut
(C) The complete verified genetic lineage and pedigree of the animal
(D) The detailed dietary history of the animal over the entire past year

2018

59. When collecting evidence for suspected malicious electrocution, which tissue must be critically examined and preserved for histopathology?
(A) Hematopoietic bone marrow extracted from the mid-shaft femur
(B) Skin and deep tissue at suspected entry and exit points
(C) Light-sensitive neural retinal tissue harvested from both eyes
(D) The epithelial lining of the urinary bladder and urethra

2019

60. Consider the following statements regarding firearm injuries in veterinary pathology:
I. The entry wound is typically smaller than the exit wound.
II. The presence of unburned gunpowder (tattooing) around a wound suggests a close-range shot.
III. Exit wounds generally display inverted edges.
IV. Recovered projectiles should be carefully handled to avoid destroying ballistic striations.
Which of the above statements is/are correct?
(A) I, II and IV
(B) I, III and IV
(C) II, III and IV
(D) I, II and III

Downloaded from www.pdf.tube

204. (B) Low serum iron, low iron-binding capacity, and high storage ferritin
The laboratory profile of anemia of chronic disease associated with neoplasia is characterized by low serum iron, low total iron-binding capacity, and normal to high storage ferritin levels. This clinical pattern is driven by inflammatory cytokines that trap iron inside macrophages and hepatocytes, making it unavailable for active erythropoiesis.

205. (A) Pancreatic beta-cell insulinoma
Hypoglycemia can occur as a paraneoplastic syndrome, most notably in patients with functional pancreatic beta-cell insulinomas. These neuroendocrine tumors autonomously secrete excessive amounts of insulin, independent of systemic glucose levels, leading to profound and persistent hypoglycemia, which clinically manifests as weakness, tremors, ataxia, and generalized epileptic seizures.

206. (B) Hamartoma
A hamartoma is a benign, tumor-like developmental malformation characterized by a disorganized mixture of mature, specialized tissues that are indigenous to the specific anatomical site of occurrence. Although they resemble neoplasms grossly and microscopically, hamartomas are non-neoplastic, growing at a rate coordinate with the surrounding normal host tissues.

207. (D) Chondrosarcoma
A chondrosarcoma is a malignant mesenchymal neoplasm characterized by the production of neoplastic cartilage by tumor cells. Microscopically, these tumors display atypical, pleomorphic chondrocytes residing within lacunae, set in a variable hyaline or myxoid cartilaginous matrix. They are locally invasive and show a moderate to high potential for metastasis.

208. (C) The histological depth of tumor invasion
For patients with localized malignant melanoma, the histological depth of tumor invasion is the most critical prognostic indicator. This parameter measures the vertical thickness of the tumor from the epidermal surface into the deeper dermis or subcutaneous layers, directly correlating with the likelihood of lymphatic invasion, metastasis, and overall survival.

209. (B) Free lipid droplets and uniform fat-filled cells with small nuclei
Cytological evaluation of a lipoma via fine needle aspiration typically yields a clean sample characterized by abundant free lipid droplets and cohesive clusters of uniform, fat-filled cells. These mature adipocytes display a large clear cytoplasm containing a single lipid vacuole that compresses the small, uniform nucleus against the cell periphery.

210. (D) Cytokeratin
Cytokeratin is the most appropriate immunohistochemical marker to confirm the epithelial origin of an anaplastic tumor. This intermediate filament protein is uniquely expressed in epithelial tissues. Identifying cytokeratin within poorly differentiated tumor cells allows pathologists to establish a diagnosis of carcinoma, ruling out mesenchymal, melanocytic, or lymphoid origins.

****

NODIA

Embedding
(B) Dehydration, Clearing, Impregnation, Embedding
(C) Impregnation, Embedding, Clearing, Dehydration
(D) Dehydration, Impregnation, Clearing, Embedding

2025
71. The primary purpose of the dehydration step in tissue processing is to:
(A) Remove stubborn residual fixative chemical precipitates and artifacts from the tissue
(B) Replace tissue water with alcohol for subsequent hydrophobic media infiltration
(C) Harden the cellular tissue structure by selectively precipitating out structural lipids
(D) Clear the cellular tissue matrix to make it highly transparent under the microscope

2016
72. Which chemical agent is most commonly utilized for the clearing step during routine tissue processing?
(A) Isopropyl alcohol
(B) Glutaraldehyde
(C) Xylene
(D) Liquid nitrogen

2021
73. What is the main advantage of using cedarwood oil instead of xylene as a clearing agent?
(A) It evaporates much faster during the paraffin wax impregnation stage.
(B) It is significantly less expensive and readily available for routine use.
(C) It simultaneously decalcifies dense bone tissue during clearing processes.
(D) It minimizes shrinkage and hardening of delicate tissues.

2010
74. Tissue embedding using paraffin wax is typically performed at a temperature slightly above the melting point of the wax. What is the standard melting point range for histological paraffin wax?
(A) 30-35°C
(B) 40-45°C
(C) 56-60°C
(D) 75-80°C

2025
75. Arrange the following tissue preparation and sectioning events in the correct operational order:
I. Knife sharpening and clearance angle adjustment
II. Paraffin block trimming
III. Floating sections on a warm water bath
IV. Picking up sections onto a glass slide
(A) I, II, III, IV
(B) II, I, IV, III
(C) II, I, III, IV
(D) I, III, II, IV

2006
76. In microtomy, which type of microtome is most frequently used for cutting routine paraffin-embedded tissue blocks?
(A) Vibrating microtome
(B) Rotary microtome
(C) Sledge microtome
(D) Ultramicrotome

2018
77. A cryostat is essentially a rotary microtome housed within a specialized freezing cabinet. At what temperature does a cryostat typically operate for cutting unfixed, frozen tissue sections?
(A) 4°C to 10°C
(B) -20°C to -30°C
(C) -70°C to -80°C
(D) -196°C

2006
78. During sectioning on a rotary microtome, consecutive tissue sections stick together edge-to-edge. This desirable outcome is known as:
(A) Chatter formation
(B) Venetian blind effect
(C) Ribbon formation
(D) Scoring

2001
79. If vertical scratches or splits appear consistently across the tissue ribbon during microtomy, what is the most likely cause?
(A) The paraffin wax used for embedding is too soft.
(B) The knife clearance angle is set significantly too high.
(C) The embedded tissue block is severely under-dehydrated.
(D) There is a nick in the microtome knife edge.

2017
80. Match the tissue processing components in List I with their corresponding chemicals in List II.
List I: (Component)
P. Primary fixative
Q. Dehydrating agent
R. Clearing agent

Click Here to Buy book on Amazon

2025

118. Algor mortis refers to the postmortem cooling of the body. The rate of cooling is mathematically modeled and depends most heavily on:
(A) The specific genetic breed and body weight of the animal
(B) The baseline blood type and metabolic rate of the animal
(C) The ambient environmental temperature and body insulation
(D) The rapid onset and baseline intensity of rigor mortis

2004

119. A condition where muscles stiffen instantly at the exact moment of death due to severe nervous system exertion prior to death is known as:
(A) Cadaveric spasm
(B) Cold shortening
(C) Autolysis
(D) Secondary flaccidity

2019

120. Which observation best differentiates a true antemortem lesion from postmortem imbibition of bile?
(A) The intense, localized yellow-green discoloration of the parenchymal tissue
(B) Its anatomical location directly adjacent to the biliary gallbladder wall
(C) Its completely uniform and diffuse distribution across the entire organ
(D) The presence of surrounding tissue inflammation and vital cell reactions

2024

121. In veterinary forensic investigations, standard necropsy protocols must be strictly followed EXCEPT when:
(A) The subject is a highly valued domestic or companion animal species
(B) Explicit deviation is required to preserve specific fragile evidence
(C) The clinical diagnosis and cause of death are already well known
(D) The owner requests a significantly faster postmortem examination

2023

122. Which fixative formulation contains picric acid, requires washing tissues in alcohol to remove yellow discoloration, and preserves glycogen well?
(A) 10% Neutral Buffered Formalin
(B) Zenker's fluid
(C) Bouin's fluid
(D) Carnoy's fluid

2014

123. Tissues fixed in Zenker's fluid require a specific post-fixation treatment to remove mercury pigment. This is typically achieved using:
(A) Concentrated solutions of alcoholic ammonia
(B) Strongly acidified dilute hydrochloric acid
(C) Prolonged running tap water washing for 24 hours
(D) Iodine treatment followed by sodium thiosulfate

2004

124. When diagnosing rabies in an animal, the preferred specimen submitted for rapid fluorescent focus inhibition testing (RFFIT) or direct fluorescent antibody (DFA) testing is:
(A) Fresh or chilled intact brain or brainstem tissues
(B) Whole systemic blood collected in EDTA tubes
(C) Formalin-fixed sections of the lobular liver
(D) Sterilely collected urine or bladder samples

2016

125. Which of the following diseases is typically a top priority for compulsory notification under Indian veterinary regulations?
(A) Severe canine parvovirus infection
(B) Highly Pathogenic Avian Influenza
(C) Chronic bovine mastitis infection
(D) Feline calicivirus complex disease

NODIA

2025

126. A necropsy reveals severe, diffuse icterus, "nutmeg" liver, and petechial hemorrhages on the epicardium. The presence of icterus is fundamentally an accumulation of:
(A) Hemosiderin
(B) Bilirubin
(C) Melanin
(D) Lipofuscin

2016

127. Which pathological term describes a localized area of ischemic necrosis within a tissue or organ, produced by occlusion of either its arterial supply or its venous drainage?
(A) Infarct
(B) Abscess
(C) Granuloma
(D) Hamartoma

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

2007

90. Match the special stains in List I with their primary target substances in List II.

List I: (Special Stain)

P. Periodic Acid-Schiff (PAS)
Q. Oil Red O
R. Alcian Blue
S. Congo Red

List II: (Target Substance)

1. Lipids
2. Amyloid
3. Acid mucins
4. Glycogen and neutral mucins

(A) P-1, Q-4, R-2, S-3
(B) P-4, Q-1, R-3, S-2
(C) P-3, Q-2, R-4, S-1
(D) P-2, Q-3, R-1, S-4

2015

91. The Periodic Acid-Schiff (PAS) reaction is widely used to demonstrate carbohydrates. What color indicates a positive PAS reaction?
(A) Black
(B) Magenta
(C) Bright blue
(D) Yellow

2019

92. To definitively prove that a PAS-positive substance in the liver is glycogen and not another carbohydrate, the tissue section should be pre-treated with which enzyme?
(A) Hyaluronidase
(B) Diastase (Amylase)
(C) Pepsin
(D) Trypsin

2013

93. Which stain must be performed on unfixed or minimally fixed frozen sections because routine processing removes the target substance?
(A) Sudan Black B for lipids
(B) Masson's Trichrome for collagen
(C) Alcian blue for acid mucins
(D) Gram's stain for bacteria

2005

94. The diagnostic target of the Ziehl-Neelsen stain is the cell wall of mycobacteria, which is rich in:
(A) Peptidoglycan
(B) Chitin
(C) Mycolic acid
(D) Lipopolysaccharide

2009

95. Consider the following statements regarding histochemical stains for pigments and minerals:

I. Perls' Prussian blue stain is used to detect hemosiderin (ferric iron).
II. Von Kossa stain is utilized to identify the presence of calcium deposits.
III. Masson-Fontana stain is used to demonstrate melanin.

Which of the above statements is/are correct?
(A) I and II
(B) I and III
(C) II and III
(D) I, II and III

2002

96. Warthin-Starry silver impregnation is the histochemical stain of choice for demonstrating which type of organism in tissue sections?
(A) Fungi
(B) Acid-fast bacilli
(C) Spirochetes
(D) Gram-positive cocci

2017

97. If a tissue section requires the demonstration of fungal hyphae and spores with high contrast, the most appropriate specialized stain is:
(A) Perls' Prussian blue
(B) Grocott's Methenamine Silver (GMS)
(C) Von Kossa
(D) Oil Red O

2018

98. Histoenzymology focuses on localizing enzyme activity in tissues. To successfully demonstrate active enzymes, the tissue must generally be:
(A) Fixed in 10% NBF for 48 hours and embedded in paraffin blocks
(B) Boiled at high temperatures to unmask potential enzymatic active sites
(C) Cleared completely in organic xylene solvents for extended time periods
(D) Preserved as fresh frozen sections without harsh fixation or heat

2006

99. The principle of simultaneous coupling in enzyme histochemistry relies on the enzyme cleaving a substrate to release a product that immediately reacts with a diazonium salt to form a visible:
(A) Azo dye
(B) Gas bubble
(C) Fluorescent tag
(D) Metallic silver deposit

Downloaded from www.pdf.tube

(B) Classical Rinderpest Virus
(C) Foot-and-Mouth Disease Virus
(D) Neurotropic Rabies Virus

2001

139. During a poultry necropsy, you observe white, chalky deposits on the epicardium, liver surface, and within the joints. This gross finding is diagnostic for:
(A) Acute systemic colibacillosis
(B) Septicemic fowl cholera disease
(C) Invasive pulmonary aspergillosis
(D) Visceral and articular gout

2005

140. Which specific condition in poultry presents with grossly enlarged peripheral nerves (e.g., sciatic, brachial plexus) that appear grayish and lose their normal striations?
(A) Marek's disease
(B) Newcastle disease
(C) Infectious Coryza
(D) Avian Encephalomyelitis

2006

141. A veterinary health certificate for export must strictly adhere to the guidelines set by which international organization?
(A) World Health Organization (WHO) of the United Nations
(B) World Organisation for Animal Health (WOAH)
(C) Food and Agriculture Organization (FAO) of the UN
(D) Centers for Disease Control and Prevention (CDC) agency

2010

142. The process of preparing bone tissue for routine microtomy requires which crucial step after fixation and before processing?
(A) Depigmentation
(B) Decalcification
(C) Desiccation
(D) Saponification

2024

143. Which chemical agent is commonly employed as a rapid decalcifying solution for dense cortical bone?
(A) Nitric acid
(B) EDTA (Ethylene diamine tetraacetic acid)
(C) Formic acid
(D) Sodium hydroxide

2011

144. In forensic toxicology, what is the primary advantage of preserving tissues in a saturated saline solution rather than formalin?
(A) Saline immediately neutralizes all pathogenic bacterial organisms
(B) Saline preserves fine micro-anatomy for histopathological slides
(C) Saline prevents chemical reaction or leaching of toxic compounds
(D) Saline significantly reduces specimen volume and shipping weight

2012

145. A magisterial inquest is mandatory under Indian law for unnatural deaths. In such vetero-legal cases, the veterinarian acts as:
(A) The lead investigative officer directing police and legal teams
(B) The official legal prosecutor representing state-level cases
(C) The final presiding judge determining absolute guilt or innocence
(D) An expert witness providing medical and pathological evidence

2007

146. Which term describes the postmortem settling of blood that results in a reddish-purple discoloration of the dependent parts of the body?
(A) Algor mortis
(B) Rigor mortis
(C) Livor mortis
(D) Pallor mortis

2014

147. When recording the consistency of an organ during necropsy, a "rubbery" texture of the lungs that fail to collapse is highly indicative of:
(A) Acute severe pulmonary congestion
(B) Interstitial pneumonia or emphysema
(C) Suppurative bronchopneumonia
(D) Congenital pulmonary hypoplasia

2013

148. A definitive etiological diagnosis of Rabies requires which specialized postmortem testing procedure on brain tissue?
(A) Routine histopathology searching for Negri bodies
(B) Bacterial cultivation on standard nutrient agar
(C) Histochemical analysis using periodic acid-Schiff
(D) Fluorescent antibody testing on brain smears

NODIA

(B) Uranyl acetate
(C) Lead citrate
(D) Phosphotungstic acid

2019

109. What is a critical limitation of glutaraldehyde that dictates the size of tissue samples prepared for electron microscopy?
(A) It dissolves bone tissue rapidly.
(B) It has a very slow rate of penetration into tissues.
(C) It evaporates instantly at room temperature.
(D) It heavily reacts with endogenous pigments blocking electron beams.

2016

110. Match the electron microscopy steps/materials in List I with their primary functions in List II.
List I: Steps/materials
P. Epoxy resin (e.g., Araldite)
Q. Ultramicrotome
R. Lead citrate
S. Diamond knife
List II: Primary functions
1. Embedding medium providing extreme hardness
2. Cutting ultrathin sections (50-90 nm)
3. Heavy metal stain for electron contrast
4. Precision cutting tool for ultrathin sectioning
(A) P-2, Q-1, R-4, S-3
(B) P-1, Q-2, R-3, S-4
(C) P-3, Q-4, R-2, S-1
(D) P-4, Q-3, R-1, S-2

2004

111. Sections cut for Transmission Electron Microscopy (TEM) are incredibly thin. What is the standard thickness range for these ultrathin sections?
(A) 1 - 5 micrometers (μm)
(B) 10 - 20 micrometers (μm)
(C) 50 - 90 nanometers (nm)
(D) 1 - 5 millimeters (mm)

2024

112. Unlike light microscopy which uses glass slides, ultrathin sections for TEM are mounted onto:
(A) Quartz coverslips
(B) Nitrocellulose membranes
(C) Copper grids
(D) Silicon wafers

2016

113. Which heavy metal salt is frequently used as a contrast-enhancing counterstain alongside uranyl acetate in TEM sample preparation?
(A) Silver nitrate
(B) Potassium permanganate
(C) Ferric chloride
(D) Lead citrate

2011

114. Scanning Electron Microscopy (SEM) is distinct from TEM primarily because SEM is designed to visualize:
(A) The detailed intracellular organelle structures in thin cross-section
(B) The internal double-stranded molecular structure of genomic DNA
(C) The three-dimensional surface topography of a bulk specimen
(D) The dynamic motion of live, unstained, fully hydrated cells

2003

115. Read the given assertion and reason carefully, and select the correct option.
Assertion (A): Critical Point Drying (CPD) is an essential step in preparing biological samples for SEM.
Reason (R): CPD allows the transitional fluid to change from a liquid to a gas without creating surface tension forces, thereby preventing the collapse of delicate surface structures.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2021

116. In Scanning Electron Microscopy, what is the specific purpose of the sputter coating process using gold-palladium?
(A) To chemically fix and stabilize the delicate biological tissue surface
(B) To rapidly decalcify and soften embedded microscopic bone spicules
(C) To selectively stain intracellular proteins for elemental analysis
(D) To render non-conductive specimen surfaces electrically conductive

2009

117. Which of the following is NOT an early postmortem change?
(A) Putrefaction
(B) Algor mortis
(C) Livor mortis
(D) Rigor mortis

Click Here to Buy book on Flipkart

S. Impregnation medium

List II: (Chemical)

1. Paraffin wax
2. Ethyl alcohol
3. 10% Neutral Buffered Formalin
4. Xylene

(A) P-3, Q-2, R-4, S-1
(B) P-2, Q-3, R-1, S-4
(C) P-3, Q-4, R-2, S-1
(D) P-1, Q-2, R-4, S-3

2008

81. In the routine Hematoxylin and Eosin (H&E) staining protocol, what is the specific role of the "bluing" agent (e.g., dilute ammonia water)?
(A) To destain any excess eosin molecules from the tissue cytoplasm
(B) To convert red soluble hematoxylin into an insoluble blue lake
(C) To act as an essential mordant to link dye molecules to the tissue
(D) To thoroughly dehydrate the section before applying the slide coverslip

2009

82. Which statement accurately describes the staining affinity of Eosin Y in routine H&E staining?
(A) It is a basic dye that stains highly acidic components such as nucleic acids pink.
(B) It selectively binds to neutral lipids in the tissue, imparting a deep red color.
(C) It relies strictly on a heavy metal chemical mordant to bind to cell membranes.
(D) It is an acidic dye that stains basic cytoplasmic proteins pink to red.

2017

83. DPX is a widely used synthetic mounting medium. What does the "X" in DPX stand for?
(A) Xylene
(B) Xylan
(C) Xanthine
(D) Xylose

2002

84. Which of the following is a primary requirement for a permanent mounting medium used in histopathology?
(A) It must readily dissolve in water.
(B) It should slowly crystallize over time to harden the slide.
(C) Its refractive index must be close to that of glass ($\sim$ 1.52).
(D) It must react with the tissue to enhance the stain intensity.

NODIA

2002

85. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): Progressive hematoxylin staining methods do not require an acid alcohol differentiation step.
Reason (R): In progressive staining, the tissue is left in the dye only until the desired intensity is reached, rather than being overstained and selectively destained.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2021

86. Which special stain is specifically utilized to differentiate collagen from smooth muscle fibers in tissue sections?
(A) Periodic Acid-Schiff (PAS)
(B) Congo Red
(C) Ziehl-Neelsen
(D) Masson's Trichrome

2013

87. In a tissue section stained with Masson's Trichrome, what color does mature collagen typically exhibit?
(A) Black
(B) Blue or green
(C) Red
(D) Yellow

2003

88. Van Gieson stain is classically used for demonstrating connective tissue. It consists of a mixture of which two primary dyes?
(A) Hematoxylin and Eosin
(B) Toluidine blue and Fast green
(C) Picric acid and Acid Fuchsin
(D) Crystal violet and Iodine

2025

89. When viewed under polarized light, amyloid deposits stained with Congo Red characteristically exhibit:
(A) Complete opacity
(B) Red metachromasia
(C) Golden-yellow fluorescence
(D) Apple-green birefringence

2018

128. When examining the heart during necropsy, determining the ratio of the right ventricular wall thickness to the left ventricular wall thickness is important. The normal ratio in a healthy adult dog is approximately:
(A) 1:10
(B) 1:3
(C) 1:1
(D) 3:1

2024

129. While performing an equine necropsy, a massive torsion of the large colon is observed. This mechanical obstruction leads to severe vascular compromise primarily described as:
(A) Severe hemorrhagic infarct
(B) Typical white/anemic infarct
(C) Simple intestinal intussusception
(D) Acute strangulating obstruction

2011

130. A tissue processing error results in the center of a paraffin block being soft and smelling strongly of xylene. What was the most likely cause?
(A) Over-dehydration
(B) Excessive clearing time
(C) Incomplete impregnation with paraffin wax
(D) Using absolute alcohol instead of 70% alcohol initially

2017

131. The primary purpose of utilizing a vacuum during the paraffin wax impregnation step is to:
(A) Significantly accelerate the cellular dehydration process
(B) Efficiently pull out the clearing agent from the tissue
(C) Safely lower the temperature and melting point of wax
(D) Successfully prevent tissues from floating on the surface

2011

132. During H&E staining, you notice the nuclei are very faintly stained blue, but the cytoplasm is a brilliant pink. Which troubleshooting step would best resolve this in future batches?
(A) Prolong exposure to Eosin or increase dye concentration
(B) Reduce exposure to the bluing agent during processing
(C) Increase concentration and activity of the acid alcohol
(D) Increase Hematoxylin staining time or replace exhausted dye

2016

133. Which of the following is considered an exogenous tissue pigment?
(A) Carbon (Anthracosis)
(B) Melanin
(C) Hemosiderin
(D) Lipofuscin

2012

134. Lipofuscin, often referred to as the "wear and tear" pigment, is typically observed in aging animals within cells of the:
(A) Epidermis
(B) Bone marrow
(C) Heart and liver
(D) Respiratory epithelium

2016

135. When formulating a morphological diagnosis, the term "suppurative" or "purulent" indicates the prominent presence of which inflammatory cell type?
(A) Neutrophils
(B) Lymphocytes
(C) Macrophages
(D) Eosinophils

NODIA

2006

136. The presence of numerous eosinophils in a tissue section, causing severe inflammation, is most characteristically associated with which type of etiology?
(A) Acute systemic viral infection
(B) Localized bacterial infection
(C) Degenerative prion disease
(D) Parasitic or allergic reaction

2003

137. Which of the following gross lesions is pathognomonic for Contagious Ecthyma (Orf) in sheep and goats?
(A) Massive, acute enlargement and congestion of the splenic parenchyma
(B) Proliferative, scabby, pustular lesions of the lips and muzzle
(C) Multifocal caseous and calcified nodules in lungs and lymph nodes
(D) Ulcerative lesions restricted to coronary bands of the hooves

2010

138. The "tiger heart" or "tiger striping" lesion, characterized by pale linear streaks of necrosis in the myocardium, is a classic finding in young calves infected with:
(A) Bovine Viral Diarrhea Virus

2019

100. Consider the following statements regarding enzyme histochemistry:

I. Alkaline phosphatase activity is optimally demonstrated at a pH of 4.5.

II. Dehydrogenases are commonly demonstrated using tetrazolium salts.

III. Esterase stains can be used to identify specific leukocyte populations, such as mast cells.

IV. Heat fixation enhances the activity of most hydrolytic enzymes.

Which of the above statements is/are correct?

(A) I and IV
(B) II and III
(C) I, II and III
(D) II, III and IV

2006

101. In diagnostic immunohistochemistry (IHC), what is the primary purpose of the "Antigen Retrieval" step?

(A) To break methylene bridges from formalin fixation and unmask epitopes
(B) To link the primary antibody to the secondary antibody or chromogen complex
(C) To digest the entire tissue section, leaving only the primary target antigen
(D) To block any active endogenous peroxidase enzymes present in red cells

2017

102. Which technique uses heat, such as microwaving or pressure cooking in a citrate buffer, to expose hidden epitopes in IHC?

(A) Proteolytic Induced Epitope Retrieval (PIER)
(B) Avidin-Biotin Complex (ABC) masking
(C) Enzymatic blocking
(D) Heat Induced Epitope Retrieval (HIER)

2021

103. Prior to applying the primary antibody in an indirect IHC protocol utilizing horseradish peroxidase (HRP), the tissue is treated with dilute hydrogen peroxide. What does this achieve?

(A) It permanently fixes the structural tissue elements to the charged glass slide.
(B) It quenches endogenous peroxidases to prevent non-specific background staining.
(C) It activates the 3,3'-Diaminobenzidine (DAB) chromogen mixture prematurely.
(D) It provides a highly selective, contrasting nuclear counterstain for the cells.

2015

104. In immunohistochemistry, when 3,3'-Diaminobenzidine (DAB) is used as the chromogen, a positive antigen-antibody reaction appears as what color?

(A) Blue
(B) Red
(C) Brown
(D) Green

2001

105. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).

Assertion (A): In direct immunofluorescence, the primary antibody is directly conjugated with a fluorochrome.

Reason (R): This method is more sensitive and provides greater signal amplification than indirect immunofluorescence.

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2010

106. Which of the following is considered an absolute necessity when preparing slides for immunofluorescence microscopy?

(A) Applying a xylene-based permanent mounting medium
(B) Staining background tissue heavily using eosin dye
(C) Applying an aqueous mounting medium to prevent quenching
(D) Utilizing high-intensity brightfield illumination lamps

2003

107. Which specialized fixative is universally preferred as the primary fixative for Transmission Electron Microscopy (TEM) due to its superior ability to cross-link proteins rapidly?

(A) 10% Neutral Buffered Formalin
(B) Bouin's fluid
(C) Zenker's fluid
(D) Glutaraldehyde

2015

108. In electron microscopy preparation, following primary fixation, a secondary fixative is applied to specifically preserve and contrast cellular lipids. This secondary fixative is:

(A) Osmium tetroxide

Downloaded from www.pdf.tube

2002
149. The legal term used to describe the intentional and cruel killing or maiming of an animal using poisons, weapons, or other destructive means is:
(A) Malicious injury
(B) Poaching
(C) Mischief
(D) Culling

2021
150. A carcass exhibiting dark, tarry, uncoagulated blood exuding from natural orifices should NOT be opened under field conditions due to high suspicion of:
(A) Rabies
(B) Brucellosis
(C) Anthrax
(D) Tuberculosis

2023
151. Which component of the triple packaging system bears the biohazard symbol, the UN number, and the shipper/consignee details?
(A) The outer packaging
(B) The primary receptacle
(C) The secondary packaging
(D) The absorbent material

2017
152. Glutaraldehyde provides excellent ultrastructural preservation for TEM because it contains two reactive:
(A) Carboxyl groups
(B) Hydroxyl groups
(C) Amino groups
(D) Aldehyde groups

2003
153. What artifact is produced when biological samples are dried in ambient air for SEM without using Critical Point Drying?
(A) Artificial and dense heavy metal crystallite precipitation
(B) Structural collapse and shrinkage from surface tension
(C) Severe premature electron beam charging during sample imaging
(D) Widespread formalin pigment artifact deposition in tissues

2018
154. Which histochemical stain demonstrates mucin, a secretion of epithelial goblet cells, most effectively?
(A) Von Kossa
(B) Masson's Trichrome
(C) Mucicarmine
(D) Sudan Black B

2017
155. The Avidin-Biotin Complex (ABC) method in immunohistochemistry is favored because of its:
(A) Low production cost and straightforward protocol execution
(B) Strong chemical cross-linking properties within tissues
(C) Complete independence from primary antibody requirements
(D) High sensitivity from the strong affinity of avidin and biotin

2014
156. In enzyme histochemistry, demonstrating esterase activity in tissue sections is particularly useful for identifying:
(A) Mast cells and monocytes
(B) Erythrocytes
(C) Smooth muscle cells
(D) Hepatocytes

2012
157. Which fixative is specifically recommended for preserving tissues intended for the demonstration of urate crystals (gout)?
(A) 10% Neutral Buffered Formalin
(B) Absolute alcohol
(C) Bouin's fluid
(D) Zenker's fluid

2024
158. In the event of an outbreak of a highly contagious exotic disease, the "stamping out" policy generally refers to:
(A) Routine vaccination of all susceptible regional animal herds
(B) Immediate slaughter of infected and in-contact susceptible animals, followed by safe carcass disposal
(C) Isolation of clinically ill animals for supportive treatment
(D) Closing regional boundaries to international transport lanes

2007
159. When collecting intestinal segments for histopathology, it is crucial to avoid scraping the mucosal surface to prevent:
(A) Loss of the delicate epithelial lining and villi
(B) Extreme tissue over-fixation by formalin agents
(C) Rapid autolytic degradation of the deep submucosa

NODIA

(D) Excessive bacterial overgrowth within mucosal crypts

2001

160. A "bread and butter" appearance of the pericardium is a classic macroscopic description of which pathological process?
(A) Purulent pericarditis
(B) Hemopericardium
(C) Fibrinous pericarditis
(D) Hydropericardium

2003

161. An infarct in the kidney or spleen typically appears grossly as a:
(A) Diffuse, dark red, and severely swollen tissue zone
(B) Circular, umbilicated, and black necrotic nodules
(C) Linear, greenish discoloration along cortical tracts
(D) Wedge-shaped, pale area with its base at the capsule

2018

162. Which organ should be evaluated first when performing a systemic examination of the thoracic cavity in an unweaned calf to assess immune status?
(A) Spleen
(B) Lungs
(C) Thymus
(D) Heart

2024

163. The primary reason for tying off (ligating) the esophagus and the rectum during necropsy of a ruminant is to:
(A) Measure the exact total length of the tract
(B) Prevent leakage of ingesta and contamination
(C) Maintain internal luminal gas for pathology
(D) Facilitate clean extraction of the pluck organs

2023

164. What is the standard diagnostic protocol for confirming a suspected case of bovine spongiform encephalopathy (BSE) postmortem?
(A) Culturing cerebral spinal fluid for pathogenic bacteria
(B) Polymerase chain reaction (PCR) on spinal fluid samples
(C) Histochemical lipid staining within cerebellar tissues
(D) Immunohistochemistry or ELISA on the brainstem obex

2019

165. During microtomy, if the clearance angle between the knife and the tissue block is too small (e.g., 0 degrees), the result is typically:
(A) The tissue block face is compressed and sections skip
(B) Sections shatter showing a venetian-blind like effect
(C) The tissue block falls completely out of the chuck
(D) The metal knife edge breaks under structural pressure

2009

166. A "sledge microtome" is primarily utilized for sectioning:
(A) Extremely tiny needle biopsies of tissue
(B) Ultrathin sections needed for transmission electron beams
(C) Very large or hard tissue blocks like whole brain or bone
(D) Frozen sections utilized during intraoperative consults

2025

167. Which of the following conditions is most likely to produce pathognomonic "Negri bodies" in the cytoplasm of neurons?
(A) Rabies
(B) Canine Distemper
(C) Pseudorabies
(D) Scrapie

2008

168. Which descriptive term is appropriate for a lesion that is flat, discolored, and non-palpable, measuring less than 1 cm in diameter?
(A) Papule
(B) Tumor
(C) Plaque
(D) Macule

2010

169. What is the fundamental principle of an abrasion and a laceration in vetero-legal pathology?
(A) Abrasions involve deep tissue tearing; lacerations are superficial epidermal scrapes
(B) Abrasions are superficial friction scrapes; lacerations are irregular blunt force tears
(C) Abrasions are caused exclusively by firearms; lacerations are caused by thermal injury
(D) There is no structural difference; the two terms are entirely synonymous in usage

Click Here to Buy book on Amazon

2024

170. Which of the following fluids serves as a transitional fluid in Critical Point Drying for SEM, directly replacing the dehydration fluid (e.g., ethanol) before transitioning to liquid carbon dioxide?
(A) Pure distilled deionized water
(B) Neutral buffered formalin fluid
(C) Amyl acetate
(D) Concentrated glutaraldehyde gas

2007

171. In immunohistochemistry, what is the role of a secondary antibody?
(A) It binds directly to the specific target antigen in tissues
(B) It reacts directly with the chromogen substrates to form dye
(C) It blocks non-specific background staining from peroxidases
(D) It binds to the primary antibody and carries the enzyme label

2002

172. When describing a pathological finding as "catarrhal," you are indicating the presence of excessive:
(A) Mucus
(B) Fibrin
(C) Blood
(D) Pus

2018

173. Severe, generalized edema resulting in fluid accumulation in multiple body cavities and subcutaneous tissues is termed:
(A) Ascites
(B) Anasarca
(C) Hydrothorax
(D) Hydropericardium

2004

174. The "Swiss cheese" brain appearance, characterized by prominent vacuolation of the neuropil under the microscope, is the hallmark lesion of:
(A) Diffuse acute viral encephalitis
(B) Transmissible spongiform encephalopathy
(C) Acute suppurative bacterial meningitis
(D) Chronic granulomatous fungal infection

2023

175. Which sectioning instrument utilizes a glass or diamond knife and operates with an advance mechanism moving in nanometer increments?
(A) Ultramicrotome
(B) Rotary microtome
(C) Sledge microtome
(D) Standard cryostat

NODIA

2013

176. What is the recommended method for fixing and preserving an entire small rodent (e.g., a mouse) if full-body perfusion is not possible?
(A) Placing the intact biological carcass directly into a freezer
(B) Injecting 10% formalin exclusively into major muscle masses
(C) Opening major body cavities and immersing the carcass in 10% NBF
(D) Desiccating the specimen with dry salt and chemical powders

2004

177. A morphological diagnosis of "diffuse, subacute, non-suppurative encephalitis" is highly suggestive of which category of infectious agent?
(A) Parasitic
(B) Bacterial
(C) Fungal
(D) Viral

2014

178. To safely package a liquid diagnostic specimen, the primary receptacle must be:
(A) Wrapped completely in thick layers of protective aluminum foil
(B) Constructed exclusively of high-density shatterproof glass
(C) Watertight, leak-proof, and wrapped in absorbent material
(D) Vented specifically to release internal gases during transit

2025

179. In electron microscopy, the source of illumination is an electron beam. What type of lenses are used to focus this beam?
(A) High-index glass lenses
(B) Electromagnetic lenses
(C) Quartz crystal lenses
(D) Diamond lenses

2014

180. The presence of a "goose-stepping" gait clinically and severe Wallerian degeneration in the peripheral nerves histopathologically is classic for which nutritional deficiency in swine?
(A) Essential vitamin A
(B) Essential vitamin E
(C) Mineralized calcium
(D) Pantothenic acid

(C) The pelvic flexure of the large colon
(D) The terminal portion of the rectum

2006

203. During an outbreak of Avian Influenza, what is the primary role of the veterinary pathologist conducting field necropsies?
(A) Curing infected birds with therapeutic antivirals
(B) Recommending alternative commercial feed mixtures
(C) Issuing soundness certificates for surviving birds
(D) Collecting samples to confirm strain for containment

2019

204. "Sausage-like" thickening of the intestinal mucosa with deep, transverse corrugations in cattle is the classic macroscopic lesion for:
(A) Johne's disease (Paratuberculosis)
(B) Bovine Viral Diarrhea infection
(C) Acute Rinderpest viral disease
(D) Classic systemic Salmonellosis

2012

205. If you observe a "hairball" completely occluding the pylorus in a calf, the correct pathological term for this concretion is a:
(A) Phytobezoar
(B) Trichobezoar
(C) Enterolith
(D) Coprolith

2004

206. When interpreting necropsy findings of a toxicological nature, the finding of bright cherry-red blood and tissues is highly suggestive of:
(A) Nitrate or nitrite compound toxicity
(B) Carbon monoxide or cyanide poisoning
(C) Chronic, cumulative lead metal poisoning
(D) Accidental ingestion of warfarin poison

2006

207. A characteristic gross lesion of nitrate/nitrite poisoning in cattle is:
(A) Chocolate-brown colored blood
(B) Bright, cherry-red colored blood
(C) Dull, greenish-colored liver tissue
(D) Diffuse renal cortical tissue necrosis

2009

208. During a forensic necropsy involving a suspected gunshot wound, the pathologist notes a wound with abraded, inward-beveled margins. This identifies the wound as:
(A) An exit wound
(B) A ricochet injury
(C) An entry wound
(D) A postmortem artifact

2022

209. To accurately identify a suspected lipid storage disease in the liver, which handling procedure is absolutely essential for the liver tissue?
(A) It must be immediately fixed in absolute alcohol to preserve the lipid
(B) It must undergo routine xylene clearing followed by paraffin embedding
(C) It must be boiled in a citrate buffer solution for antigen retrieval
(D) It must be frozen unfixed or formalin-fixed for cryostat sectioning

2008

210. Under scanning electron microscopy (SEM), backscattered electrons provide primary information about the specimen's:
(A) Detailed and fine surface topographical shape
(B) Internal crystalline lattice structure design
(C) Composition and atomic number differences
(D) Specimen fluid dynamics observed in real-time

****

Downloaded from www.pdf.tube

(D) Gravitational hypostatic congestion

2012

193. The microscopic appearance of "heart failure cells" in the lungs of a dog with chronic left-sided heart failure refers to:
(A) Lymphocytes infiltrating mucosal surfaces
(B) Multinucleated giant inflammatory cells
(C) Eosinophils migrating through bronchioles
(D) Hemosiderin-laden alveolar macrophages

2009

194. In the triple packaging system for shipping biological samples, the documentation (e.g., submission forms) should be placed:
(A) Inside the primary receptacle directly alongside the tissue specimen
(B) Inside the secondary packaging, sealed tightly in a biohazard bag
(C) Between the secondary and outer packaging in a waterproof pouch
(D) Taped directly onto the exterior surface over the UN designation

2006

195. Which fixative agent acts simultaneously as both a fixative and a strong decalcifying agent?
(A) Bouin's fluid
(B) Perenyi's fluid
(C) Glutaraldehyde
(D) 10% Neutral Buffered Formalin

2017

196. The condition known as "osteomalacia" is characterized grossly by:
(A) Excessive, generalized thickening and hardening of bone trabeculae
(B) Complete and irreversible destruction of key articular cartilages
(C) Rapid, uncontrolled neoplastic proliferation of bone osteoblasts
(D) Soft, pliable bones in adults due to deficient osteoid mineralization

2017

197. The presence of multifocal, caseous, calcified nodules in the lungs and lymph nodes of cattle is highly indicative of:
(A) Bovine Tuberculosis
(B) Contagious Bovine Pleuropneumonia
(C) Pasteurellosis
(D) Dictyocaulosis

2018

198. Which of the following is considered an indication for issuing an official health certificate for an animal?
(A) The animal requires immediate clinical euthanasia
(B) The animal is showing signs of scheduled disease
(C) The animal is being transported across borders
(D) The owner requests a refund on health insurance

2018

199. When identifying lesions associated with suspected starvation or severe malnutrition, the most consistent gross postmortem finding is:
(A) Serous atrophy of fat in the coronary groove and bone marrow
(B) Severe, widespread hepatic lipidosis throughout the liver lobes
(C) Moderate to severe accumulation of ascites and fluid in thorax
(D) Diffuse and acute mucosal hemorrhages throughout the stomach

NODIA

2025

200. In immunohistochemistry, an internal positive control is defined as:
(A) A completely separate tissue slide known to lack target antigen
(B) The deliberate, controlled omission of primary antibody steps
(C) Using secondary antibodies raised against different host species
(D) Normal tissue components within the patient's section that naturally express the target antigen

2018

201. The pathological descriptor "fibrinous" refers to:
(A) Chronic scar tissue formation with collagen bundles
(B) Acute exudation of fibrinogen forming fibrin networks
(C) Accumulation of pus and necrotic cellular debris
(D) Pathological, excessive production of glandular mucin

2012

202. The most common site for impaction in the equine gastrointestinal tract, often leading to fatal colic, is the:
(A) The cardiac sphincter of the stomach
(B) The descending portion of the duodenum

9. (D) Hemoglobin imbibition diffuses into tissues without vascular rupture or inflammation.
Postmortem staining of tissues occurs when red blood cells lyse and release hemoglobin, which passively diffuses through vessel walls into adjacent structures. This process is easily distinguished from antemortem hemorrhage because it lacks any localized inflammatory tissue response, fibrin deposition, or intact, extravasated erythrocytes within the affected interstitial spaces during microscopic examination.

10. (B) P-2, Q-4, R-1, S-3
Postmortem changes follow distinct physical and chemical pathways. The cooling of the carcass matches the surrounding environmental temperature, while bacterial gas reactions produce iron sulfide pigments that discolor tissues. Gravitational forces pull blood down into lower capillary beds, and the progressive exhaustion of adenosine triphosphate halts the relaxation phase of muscle fibers.

11. (B) Left lateral recumbency
Positioning a large ruminant carcass on its left side is the standard diagnostic protocol because it places the massive, gas-filled rumen underneath the other visceral organs. This orientation allows the prosector to make an incision on the right abdominal wall, providing an unobstructed view and easy access to the intestines, liver, and kidneys.

12. (B) To allow the large colon and cecum (situated primarily on the right) to be easily accessed and exteriorized
Placing an equine carcass on its left side provides the optimal pathway for evaluating the massive gastrointestinal tract. This positioning places the extensive cecum and large colon uppermost on the right side of the abdominal cavity, allowing the prosector to easily manipulate, unpack, and thoroughly examine these voluminous organs without tearing the delicate mesentery.

13. (A) Decalcifying the entire skull
The initial external assessment focuses on evaluating the carcass before making internal incisions. This includes checking the nutritional state, examining the skin, hair, and hooves, and inspecting all natural body openings for abnormal discharges or lesions. Mineral extraction of the skull bone is a specialized laboratory procedure performed much later during tissue preparation.

14. (C) Tongue, larynx, trachea, lungs, and heart
The extraction of the pluck involves removing the continuous anatomical structures of the upper respiratory and cardiovascular systems. By removing the tongue, larynx, trachea, esophagus, lungs, and heart as a single, intact unit, the pathologist can systematically examine the entire respiratory and cranial digestive pathways for congenital anomalies, systemic infections, or localized inflammatory lesions.

15. (C) I only
To prevent extensive contamination of the abdominal cavity and other visceral organs with ingesta, the rumen of a ruminant must be opened and emptied only after all other abdominal organs have been thoroughly evaluated and removed. This sequence maintains sanitary conditions and prevents masking of subtle mucosal lesions in other digestive segments.

16. (C) It preserves the external carcass appearance for owner viewing or burial.
This specialized postmortem procedure is designed to obtain crucial diagnostic tissues and examine internal organs while minimizing visible external damage. It is particularly valuable for beloved companion animals because it allows the veterinarian to address the owner's need for diagnostic closure while fully maintaining the cosmetic integrity of the carcass for subsequent viewing.

17. (B) Dorsal recumbency
Placing canine and feline patients on their backs is the standard orientation for routine small animal postmortem examinations. This symmetrical positioning allows for an equal, bilateral reflection of the skin and limbs, providing excellent, unobstructed access to both the thoracic and abdominal cavities, and facilitating a structured, systematic evaluation of all internal organs.

18. (D) Performing whole-body perfusion with a fixative via the left ventricle
To achieve rapid and uniform tissue preservation in small laboratory rodents, the vascular system is flushed and perfused with a chemical fixative prior to organ harvesting. This method ensures that the fixative reaches delicate deep tissues immediately, preventing the rapid postmortem autolysis that typically occurs in these small species due to high metabolic rates.

Click Here to Buy book on Flipkart

ANSWERS

1. (B) The complete and irreversible cessation of all vital functions of the body
Somatic death represents the permanent and complete cessation of all vital physiological functions of an organism as a whole, specifically involving the permanent loss of cardiac, respiratory, and brain activities. This systemic state precedes the subsequent gradual death of individual cells and tissues, which occurs over a varying period depending on local environmental factors and cellular metabolic rates.

2. (D) Algor mortis, livor mortis, rigor mortis
Following somatic death, the body undergoes a series of predictable physical and chemical changes. The initial phase involves the gradual cooling of the carcass to match the ambient temperature, followed by the gravitational pooling of blood in lower regions, and subsequently the progressive stiffening of muscle groups due to cellular energy depletion.

3. (A) The postmortem depletion of ATP preventing the dissociation of actin and myosin filaments
The postmortem stiffening of muscles occurs because cellular respiration ceases, stopping the production of adenosine triphosphate. Without this energy molecule, the actin and myosin filaments within muscle myofibrils remain locked in a permanent cross-linked state. This prevents muscle relaxation, which is highly dependent on active calcium transport and ATP binding for filament dissociation.

4. (C) It refers to the sequential death of individual cells and tissues following somatic death.
Molecular death represents the final stage of the dying process, involving the progressive and irreversible cessation of metabolic activity in individual cells and tissues. This process occurs hours after systemic somatic death, as different cell types have varying capacities to survive anaerobic conditions and utilize remaining intracellular energy reserves before undergoing irreversible autolytic degradation.

5. (A) Both A and R are true and R is the correct explanation of A.
Hypostatic congestion relies heavily on the physical volume of blood present within the circulatory system at the time of death. In cases of severe exsanguination or acute blood loss, the markedly reduced volume of intravascular fluid prevents significant gravitational settling of erythrocytes, resulting in highly indistinct or absent postmortem discoloration in the dependent parts of the carcass.

6. (D) It is characterized by tissue degradation by saprophytic bacteria and their enzymes.
Putrefaction is distinguished from sterile autolysis by the active involvement of anaerobic and saprophytic microorganisms, mostly originating from the gastrointestinal tract. These bacteria proliferate rapidly after death, fermenting tissues and producing characteristic gases, foul odors, and distinct pigments, whereas autolysis is purely driven by the self-digestion of tissues by endogenous lysosomal enzymes.

7. (B) Pseudomelanosis
This postmortem alteration develops when hydrogen sulfide gas produced by proliferating intestinal bacteria diffuses into surrounding tissues and reacts with iron released from degraded hemoglobin. This chemical reaction produces iron sulfide, a dark pigment that imparts a characteristic green, grey, or black discoloration to neighboring abdominal organs, particularly the liver, kidneys, and spleen.

8. (C) Postmortem clots are composed entirely of leukocytes with no erythrocyte involvement.
Postmortem blood clotting involves the natural settling of red blood cells before the coagulation process is completed, resulting in a layered appearance with a lower red portion and an upper yellowish plasma layer. Both layers contain blood cells, whereas antemortem thrombi are dry, friable, layered, and firmly attached to the damaged vascular endothelium.

Downloaded from www.pdf.tube

29. (A) Distribution
Using this specific terminology describes a lesion pattern where multiple, distinct, localized areas of disease start to touch, merge, and form larger confluent zones of tissue damage. This represents the spatial distribution of the lesions within the affected organ, indicating a progressive process that is spreading across the parenchymal tissue.

30. (B) Organ identity, size/weight, color, consistency, distribution of lesions
Systematically documenting pathological findings requires a structured approach to minimize subjectivity. The pathologist must first identify the affected organ, record its physical dimensions or mass, describe its color variations, evaluate its physical texture or consistency, and finally map the spatial pattern and distribution of any observed lesions across the tissue.

31. (D) Nodule
A nodule is defined as an elevated, solid, circumscribed lesion that extends deeper into the dermis or parenchymal tissue and is larger than a small, superficial papule. This term is routinely used in descriptive pathology to characterize localized neoplastic growths, chronic granulomatous inflammatory reactions, or hyperplasia within various organs.

32. (C) Umbilicated
An umbilicated lesion features a distinct, central depression that resembles a navel, often surrounded by a raised, firm outer border. This characteristic macroscopic appearance is frequently observed in rapidly growing neoplastic lesions undergoing central ischemic necrosis, as well as in certain viral infections that cause localized epithelial hyperplasia and subsequent central degeneration.

33. (B) Using household items like "fruit-sized" to describe lesion size
Descriptive pathology requires objective, reproducible, and standardized measurements using metric units such as centimeters or millimeters to ensure scientific accuracy. Utilizing subjective comparisons to common household items or food products introduces unnecessary variability and is considered highly inappropriate in formal diagnostic necropsy reports and vetero-legal documentation.

34. (D) A lesion characteristic of a single disease that allows immediate diagnosis
A pathognomonic lesion is a distinct, highly specific macroscopic or microscopic change that is uniquely associated with a single, particular disease entity. The presence of such a lesion provides sufficient diagnostic certainty to allow the pathologist to make an immediate, definitive diagnosis without requiring further confirmatory laboratory testing or history.

35. (A) II, III, IV, I
The diagnostic pathway in veterinary pathology begins with a detailed, objective description of all observed macroscopic lesions. These findings are synthesized to identify primary morphological changes, which are then grouped into recognized clinical syndromes or physiological disorders, ultimately leading to the identification and formulation of a specific etiological cause.

36. (C) Classical Swine Fever
The development of concentric, raised, necrotic mucosal lesions, commonly termed button ulcers, in the cecum and colon is a classic macroscopic feature of Classical Swine Fever. These lesions arise from localized thrombosis of submucosal blood vessels and subsequent ischemic necrosis of the overlying intestinal mucosa, often complicated by secondary bacterial invaders.

NODIA

37. (A) "Zebra striping" of the large intestinal mucosa
Zebra striping refers to the severe, linear congestion and hemorrhage observed along the mucosal folds of the colon and rectum. This striking macroscopic lesion is highly characteristic of acute rinderpest in cattle, resulting from viral-induced vascular damage and intense inflammation within the lower gastrointestinal tract of affected animals.

38. (D) Right side of the heart
A nutmeg liver arises from chronic passive congestion, which is primarily caused by right-sided heart failure. The elevated hydrostatic pressure in the vena cava is transmitted back to the hepatic veins, causing blood to pool in the centrilobular sinusoids, leading to congestion and necrosis that contrasts with the fatty change in periportal hepatocytes.

19. (A) The spiral colon located in the left abdominal cavity
Swine possess a unique gastrointestinal anatomy characterized by a large, cone-shaped spiral colon situated predominantly in the left half of the abdominal cavity. Recognizing this species-specific feature is essential during necropsy to avoid accidental laceration of the bowel and subsequent contamination of the peritoneal space with fecal material during organ extraction.

20. (B) II and III
Extracting the brain of a carnivore requires multiple precise bone cuts rather than a single transverse incision to preserve neural architecture. Additionally, opening the abdominal cavity first is the standard practice in mammalian necropsies to prevent displacement of the diaphragm and facilitate proper inspection of the pleural space and negative thoracic pressure.

21. (A) Gallbladder
Equines naturally lack a gallbladder, a normal anatomical variation where bile produced by the liver is secreted continuously and directly into the duodenum via the hepatic duct. Recognizing the absence of this organ is fundamental during equine postmortem examinations to avoid misidentifying it as a pathological developmental defect or surgical excision.

22. (D) To prevent aerosolization of feathers and infectious pathogens
Wetting the avian carcass in a disinfectant solution containing detergent before starting the necropsy is a critical biosafety measure. This practice prevents loose feathers, dander, and infectious airborne particles, such as those associated with zoonotic diseases like chlamydiosis or avian influenza, from contaminating the air and posing an inhalation risk to the prosector.

23. (B) Uropygial gland
The uropygial gland, or preen gland, is a bilobed sebaceous structure located dorsally at the base of the tail in most bird species. During avian postmortem examinations, reflecting the skin in this region allows the pathologist to inspect this gland for swelling, impaction, neoplasia, or inflammatory changes that could affect feather quality and waterproofing.

24. (C) Marek's Disease
Marek's disease is a common herpesviral infection in poultry characterized by the neoplastic proliferation of T-lymphocytes. A classic gross lesion of this disease is the marked, asymmetrical enlargement of major peripheral nerves, particularly the sciatic, vagus, and brachial nerves, resulting in a loss of their normal transverse striations and a dull, grayish appearance.

25. (A) I, II and IV
The avian digestive tract contains a highly specialized glandular stomach and an esophageal storage organ, while the bursa of Fabricius serves as a key dorsal lymphoid organ. Unlike mammals, birds lack a diaphragm, meaning their thoracic and abdominal cavities are continuous, and their non-expansile lungs are embedded within the dorsal thoracic wall.

26. (B) Crop → Proventriculus → Ventriculus → Small intestine
The avian digestive tract follows a highly specialized sequence designed for efficient food storage and mechanical breakdown. Ingesta moves from the esophagus into the crop for temporary storage, passes into the glandular proventriculus for enzymatic digestion, enters the muscular ventriculus for grinding, and finally proceeds into the small intestine for nutrient absorption.

27. (B) Distribution
Describing how lesions are arranged within an organ is vital for establishing an accurate morphological diagnosis. Documenting whether abnormalities are focal, multifocal, diffuse, or locally extensive provides essential clues about the underlying pathophysiology, helping the pathologist determine if the disease process is blood-borne, aerogenous, or a localized tissue injury.

28. (B) Consistency
The term friable describes a tissue that is easily crumbled, broken, or pulverized under gentle pressure. This characteristic directly informs the pathologist about the structural integrity and consistency of the organ, often indicating processes such as cellular necrosis, acute inflammation, or rapid postmortem autolysis that weaken normal cellular and extracellular matrix bonds.

Click Here to Buy book on Amazon

49. (B) Allow government mobilization of resources for containment and stamping out
Mandatory reporting of high-consequence animal diseases allows state and national authorities to quickly implement emergency response protocols. This rapid mobilization of veterinary resources, quarantine measures, and stamping-out strategies is crucial for containing highly contagious outbreaks before they can spread to wider geographic regions and cause severe economic loss.

50. (C) P-3, Q-2, R-1, S-4
Different types of diagnoses provide distinct clinical and pathological information. A morphological statement details tissue changes and severity, while an etiological term names the causative pathogen. A syndromic diagnosis describes the functional systemic failure, and a clinical assessment is based on observed clinical symptoms and history before necropsy.

51. (A) To isolate animals to observe for infectious diseases before integration
Veterinary quarantine is a fundamental biosecurity practice designed to isolate newly arrived or potentially exposed animals in a secure facility. This temporary isolation allows veterinary authorities to monitor the animals for clinical signs of infectious disease and perform necessary diagnostic testing before they are integrated into resident populations.

52. (C) Maximum incubation period of the disease of concern
The isolation period in veterinary quarantine is calculated based on the maximum incubation period of the specific infectious diseases of concern. This duration ensures that any exposed, subclinically infected animal will have sufficient time to develop clinical symptoms or test positive before the quarantine period ends, preventing disease introduction.

53. (A) Soundness and health certificate
This formal veterinary document is issued by an authorized veterinarian following a thorough physical examination of the animal. It provides legal and clinical assurance that the animal exhibits no signs of infectious or contagious diseases and is physically fit for transport, sale, or entry into another geographic jurisdiction.

54. (D) Halt all quarantine station movement and notify statutory authorities
Detecting a suspected exotic or high-consequence disease within a quarantine facility requires an immediate emergency response. All movement of animals, personnel, and materials within and out of the station must be halted immediately, and the relevant national or state animal health authorities must be notified to implement biosecurity and containment protocols.

55. (C) I and II
A vetero-legal postmortem examination is a highly formalized legal procedure that requires proper authorization from legal authorities before starting. To ensure that all findings and collected specimens are legally admissible in court, the prosector must strictly maintain the chain of custody and document all photographic evidence with scales and labels.

56. (C) Documenting uninterrupted tracking of evidence from collection to disposition
Establishing a secure chain of custody is a fundamental requirement in legal proceedings. It involves documenting the chronological possession, transfer, analysis, and storage of physical evidence collected during a necropsy, ensuring that each handler is recorded, thereby preventing any claims of tampering, contamination, or loss of evidence before trial.

57. (D) Saturated solution of common salt (NaCl)
For toxicological analysis, preserving visceral tissues in a saturated sodium chloride solution is highly recommended. This preservative prevents tissue decomposition while avoiding chemical interference with analytical toxicology assays, unlike formalin, which can chemically cross-link proteins, modify toxic compounds, and interfere with the extraction of many common heavy metals.

58. (A) Specific identification marks, brands, tags, and microchips
In insurance-related postmortem examinations, establishing the identity of the animal is of paramount legal importance. The pathologist must carefully document and record all physical identification markers, such as ear tags, brands, tattoos, coat patterns, and microchips, to confirm that the deceased animal matches the individual covered by the policy.

NODIA

39. (B) Foot-and-Mouth Disease
The formation of fluid-filled vesicles that subsequently rupture to form painful erosions on the buccal mucosa, dental pad, tongue, and coronary bands is the hallmark lesion of Foot-and-Mouth Disease. This highly contagious viral infection targets stratified squamous epithelium, causing severe cell swelling, acantholysis, and subsequent vesicular dermatitis.

40. (D) IV only
Diamond-shaped skin lesions are the classic, highly characteristic gross manifestation of Swine Erysipelas, caused by Erysipelothrix rhusiopathiae, rather than pseudorabies. These distinct cutaneous lesions are produced by localized vasculitis and subsequent thrombosis within the dermal capillaries, leading to rhombboidal areas of ischemic skin necrosis in pigs.

41. (D) Contagious Bovine Pleuropneumonia (CBPP)
Contagious Bovine Pleuropneumonia, caused by Mycoplasma mycoides subsp. mycoides, typically produces a severe, unilateral fibrinous pleuropneumonia. The characteristic marbling appearance of the lung parenchyma is caused by marked widening of the interlobular septa by serofibrinous fluid, combined with varying stages of red and gray hepatization within the pulmonary lobules.

42. (A) The organ affected, pathological process, distribution, severity, and duration
A morphological diagnosis is a structured, concise diagnostic statement that summarizes the key features of a lesion. It must include the specific organ or tissue involved, the nature of the pathological process, the spatial distribution of the changes, the severity of the lesions, and the estimated duration or chronicity of the condition.

43. (B) Causative agent responsible for the lesions
While a morphological diagnosis focuses on describing the structural changes and host tissue responses, an etiological diagnosis identifies the specific causative agent responsible for those changes. This type of diagnosis names the precise bacterium, virus, parasite, fungus, or chemical toxin that initiated the cellular damage and subsequent disease process.

44. (B) Morphological diagnosis
The phrase is a classic example of a morphological diagnosis because it systematically identifies the estimated duration, the severity of the process, the spatial distribution throughout the tissue, the specific exudative host response, and the anatomical organ affected, without naming the specific infectious or non-infectious causative agent responsible.

45. (C) A is true but R is false.
A syndromic diagnosis is a useful clinical tool that groups concurrent pathological and clinical findings into a recognized physiological disorder. However, it does not identify the specific causative pathogen directly; instead, it provides a functional assessment of organ system failure, which still requires further diagnostic laboratory testing for definitive etiological confirmation.

46. (C) To report the outbreak to the designated local or state authority
Under the statutory provisions of this national animal health legislation, any veterinarian who suspects or confirms the presence of a scheduled notifiable disease is legally obligated to immediately notify the local veterinary authority. This rapid communication is essential to initiate timely official containment measures and prevent further spread.

47. (D) Regulation of veterinary clinic registration fees
The primary focus of this infectious disease control act is to safeguard national animal health and livestock-dependent economies by preventing, controlling, and eradicating scheduled contagious diseases. The administrative regulation of private clinical fees is outside the scope of this biosecurity legislation, which focuses strictly on disease containment and quarantine.

48. (C) Endemic
In veterinary epidemiology, a disease is classified as endemic when it maintains a constant, predictable presence within a specific geographic population or region without external importation. This steady state indicates that the pathogen has established a stable equilibrium within the local environment and its susceptible host populations over time.

Downloaded from www.pdf.tube

2009
181. A "cor villosum" or "shaggy heart" appearance is typically the result of:
(A) Severe fibrinous epicarditis
(B) Acute myocardial infarction
(C) Endocardiosis of the mitral valve
(D) Ruptured chordae tendineae

2016
182. Which specialized histopathological technique allows for the rapid diagnosis of a malignancy during an ongoing surgical operation?
(A) Critical point drying techniques
(B) Paraffin embedded tissue sectioning
(C) Frozen sectioning using a cryostat
(D) High-resolution electron microscopy

2006
183. Which special stain is used to specifically demonstrate collagen fibers in histological sections, staining them brilliant blue or green?
(A) Periodic acid-Schiff (PAS)
(B) Masson's trichrome
(C) Prussian blue
(D) Von Kossa

2021
184. The pathological term "malacia" (e.g., encephalomalacia) translates to:
(A) Abnormal hardening of structural tissue
(B) Chronic inflammation of cellular tissue
(C) Rapid, abnormal growth of organ tissue
(D) Softening or liquefaction of tissue

2004
185. When examining a horse with a history of severe colic, discovering a tight twist of the small intestine around its mesenteric axis is documented as:
(A) Intussusception
(B) Volvulus
(C) Herniation
(D) Eventration

2001
186. Which chemical serves as a common mordant in Harris's Hematoxylin formulation?
(A) Iron chloride
(B) Iodine crystal
(C) Potassium alum
(D) Tungstic acid

2014
187. The term "atelectasis" describes which gross pathological condition of the lung?
(A) Alveolar hyperinflation and rupture
(B) Severe purulent parenchymal response
(C) Widespread fibrotic tissue scarring
(D) Incomplete lung expansion or collapse

2003
188. In avian pathology, the finding of nodular, proliferative, crusty skin lesions on the unfeathered areas of the head, comb, and wattles is characteristic of:
(A) Marek's Disease
(B) Infectious Laryngotracheitis
(C) Fowl Pox
(D) Avian Influenza

2004
189. "Flea-bitten" kidneys—characterized by numerous pinpoint petechial hemorrhages on the cortical surface—are a classic macroscopic lesion of which disease in swine?
(A) Porcine Epidemic Diarrhea
(B) Classical Swine Fever
(C) Swine Influenza
(D) Porcine Parvovirus

2019
190. What is the fundamental principle of fluorochrome emission in immunofluorescence microscopy?
(A) The fluorochrome selectively reflects white light based on structural density
(B) The fluorochrome absorbs shorter wavelengths and emits longer wavelengths
(C) The fluorochrome immediately releases fast electrons when hit by lasers
(D) The fluorochrome absorbs longer wavelengths and emits shorter wavelengths

2024
191. During the embedding phase of tissue processing, ensuring the correct "orientation" of the tissue in the mold is critical because:
(A) It dictates the plane of sectioning to keep key layers visible
(B) It determines the baseline chemical color of the final H&E stain
(C) It prevents the embedding wax from crystallizing too rapidly
(D) It alters the fundamental chemical composition of tissue blocks

NODIA

2022
192. Which postmortem artifact can mimic antemortem gastric ulceration or necrosis, especially in the rumen or stomach, if necropsy is delayed?
(A) Systemic postmortem blood imbibition
(B) Normal skeletal rigor mortis onset
(C) Postmortem autolysis by gastric acids

59. (B) Skin and deep tissue at suspected entry and exit points
Investigating suspected electrical injury requires locating and harvesting tissues from the electrical entry and exit sites. Histopathological examination of these skin and deep tissue samples is crucial, as they can reveal pathognomonic thermal changes, such as epidermal nuclear streaming, dermal collagen denaturation, and localized vascular thrombosis, confirming electrocution of the animal.

60. (A) I, II and IV
Ballistic injuries present characteristic macroscopic features that help determine the distance and direction of the shot. Entrance wounds typically feature inward-rolled margins and gunpowder residue if fired at close range, while exit wounds are larger and display everted edges. Recovered bullets must be handled carefully to preserve markings for forensic analysis.

61. (A) Tetanic convulsions and extreme extensor rigidity
Strychnine is a potent neurotoxin that selectively blocks glycine receptors in the spinal cord and brain, leading to loss of postsynaptic inhibition. This results in severe, uncontrolled reflex activity and generalized tetanic convulsions, which typically present as extreme extensor rigidity, opisthotonos, and rapid death due to respiratory muscle paralysis.

62. (C) Liver
The liver is the primary site of xenobiotic metabolism and biotransformation in the body, making it the most important organ to collect for toxicological analysis. It accumulates high concentrations of many toxins, heavy metals, and their metabolites, providing a reliable diagnostic tissue sample for confirming exposure to various poisons.

63. (D) To contain leaks from the primary receptacle and hold absorbent material
Under the standardized triple packaging system, the secondary container is designed to be leak-proof and durable. It surrounds the primary tissue receptacle, holding sufficient absorbent material to capture the entire volume of fluid contents in the event of primary container failure, thereby preventing leakage and contamination during diagnostic transport.

64. (C) UN 3373
The international shipping code UN 3373 is the legally mandated label for packages containing Category B infectious substances. This classification applies to biological specimens, such as tissue samples or fluids collected for diagnostic purposes, that are suspected of containing infectious pathogens but do not meet the criteria for Category A substances.

65. (B) I and V
Preserving tissues for PCR requires immediate freezing or specialized RNA stabilizers, as formalin cross-links DNA and RNA, hindering molecular extraction. Additionally, cooling elements like ice packs must be placed in the outer packaging layers to prevent direct contact with the primary specimen container, avoiding localized freezing or sample degradation.

66. (D) RNAlater
This specialized aqueous reagent is formulated to rapidly penetrate tissues and stabilize cellular RNA. It effectively protects RNA from degradation by endogenous ribonucleases, preserving the molecular integrity of the sample at room temperature or during transport, which is essential for subsequent gene expression studies and PCR-based diagnostic assays.

67. (A) The cross-linking of proteins to halt autolysis
The primary goal of tissue fixation is to preserve cellular morphology and relationships in a lifelike state. This is achieved by using chemical fixatives, such as formalin, to form covalent cross-links between amino acids in structural proteins, thereby disabling autolytic enzymes and preventing subsequent bacterial degradation of the tissue.

68. (D) 10:1 to 20:1
Achieving adequate and uniform tissue preservation requires a substantial volume of chemical fixative relative to the size of the tissue specimen. The standard recommended ratio is between ten to twenty parts fixative to one part tissue, ensuring that the active ingredients do not become depleted as they penetrate and react with the sample.

69. (A) When blood-rich tissues are fixed in unbuffered, acidic formalin
Formalin pigment is an unwanted artifact that develops when hemoglobin-rich tissues are

Click Here to Buy book on Flipkart

149. (C) Mischief
In Indian legal terminology, the intentional and unlawful destruction, killing, poisoning, or maiming of any animal is categorized under the offense of mischief. This legal classification, detailed in the Indian Penal Code, addresses acts that cause wrongful loss or damage to property, which legally includes animals, and carries specific criminal penalties for perpetrators.

150. (C) Anthrax
Anthrax is a peracute, zoonotic bacterial infection caused by Bacillus anthracis. Affected carcasses typically show incomplete rigor mortis and dark, tarry, uncoagulated blood oozing from the nostrils, mouth, and anus. Opening such a carcass is strictly prohibited because exposure to atmospheric oxygen triggers the vegetative bacteria to form highly resilient spores, contaminating the environment for decades.

151. (A) The outer packaging
The triple packaging system is used to transport infectious biological specimens safely. The system consists of three distinct layers. The primary receptacle holds the sample, and the secondary packaging provides shock protection. The outer packaging is the final rigid exterior layer that must carry all regulatory labeling, including the biohazard symbol, UN shipping numbers, and address details.

152. (D) Aldehyde groups
Glutaraldehyde is widely recognized as the premier fixative for preserving cellular ultrastructure in electron microscopy. This chemical is a dialdehyde, meaning it possesses two highly reactive aldehyde groups. These dual groups allow it to effectively cross-link adjacent amino groups on proteins, creating a highly stable and rigid macromolecular network that preserves delicate organelles.

153. (B) Structural collapse and shrinkage from surface tension
During scanning electron microscopy sample preparation, liquid must be completely removed from the specimen. If dried in ambient air, the surface tension forces exerted at the receding liquid-gas boundary are massive. This physical force causes severe structural collapse, distortion, and shrinking of delicate surface features, making critical point drying essential to bypass these destructive forces.

154. (C) Mucicarmine
Mucicarmine is a specialized histochemical stain used to identify acid mucopolysaccharides in tissue sections. The carmine dye complex selectively binds to epithelial mucins, which are secreted by goblet cells in gastrointestinal and respiratory tissues. This staining reaction colors the mucin brilliant pink or red, allowing for easy identification of mucin-producing cells and tumors.

NODIA

155. (D) High sensitivity from the strong affinity of avidin and biotin
The avidin-biotin complex method is a widely utilized immunohistochemistry technique. This method exploits the exceptionally strong, non-covalent biological affinity between avidin and biotin. This strong binding allows multiple biotinylated reporter enzymes to link to a single secondary antibody, significantly amplifying the signal at the antigen site and providing exceptional diagnostic sensitivity in tissue sections.

156. (A) Mast cells and monocytes
Enzyme histochemistry demonstrates active functional enzymes in frozen tissue sections. Staining for non-specific esterase activity is a classic technique used to identify specific leukocyte lineages. This enzyme is highly active in cells of the mononuclear phagocyte system, making it particularly useful for identifying and differentiating monocytes, macrophages, and mast cells in pathological tissue samples.

157. (B) Absolute alcohol
Urate crystals, which accumulate in visceral and articular gout, are highly soluble in water and aqueous solutions. Using standard water-based fixatives like formalin will dissolve these crystals, leaving empty spaces in the tissue. Fixing the tissue in absolute alcohol, a non-aqueous solvent, preserves the integrity of these crystals for specialized histopathological and polarization microscopy.

158. (B) Immediate slaughter of infected and in-contact susceptible animals, followed by safe carcass disposal
The stamping out policy is an aggressive disease control strategy used during outbreaks of highly contagious exotic diseases. It involves the immediate, humane slaughter of all infected and clinically susceptible in-contact animals within

80. (A) P-3, Q-2, R-4, S-1
Each step of tissue preparation relies on specific chemical reagents. Formalin is used to cross-link proteins during fixation, alcohol removes water during dehydration, xylene clears the tissue and removes alcohol, and molten paraffin wax infiltrates the tissue to provide structural support during embedding and subsequent microtomy sectioning.

81. (B) To convert red soluble hematoxylin into an insoluble blue lake
Bluing is a critical step in the H&E staining process. It involves exposing the stained tissue to an alkaline solution, which shifts the pH and converts the red, acid-soluble hematoxylin into an insoluble, dark blue coordination complex. This ensures that nuclear chromatin is sharply defined and permanently colored on the slide.

82. (D) It is an acidic dye that stains basic cytoplasmic proteins pink to red.
Eosin Y is an acidic, anionic dye that exhibits a strong binding affinity for basic, cationic components within the tissue. It targets basic cytoplasmic proteins, collagen, and red blood cells, staining them in varying shades of pink, orange, and red, which provides a sharp contrast to the blue-stained cell nuclei.

83. (A) Xylene
DPX is a widely used, rapid-drying synthetic mounting medium for histological sections. Its name is an acronym derived from its chemical components: Distrene (a synthetic plasticizer), Plasticizer (dibutyl phthalate), and Xylene (the solvent used to dissolve the components and maintain miscibility with clearing agents on the glass slide).

84. (C) Its refractive index must be close to that of glass (~1.52)
A key requirement for any permanent mounting medium is that its refractive index matches that of the glass slide and coverslip. This optical alignment prevents light scattering and distortion when the slide is viewed under the microscope, ensuring maximum clarity, high resolution, and sharp focus of the stained tissue structures (≈ 1.52).

NODIA

85. (A) Both A and R are true and R is the correct explanation of A.
Progressive staining is designed to stain tissues to a precise, desired end-point, eliminating the need for subsequent differentiation steps. The tissue is left in the hematoxylin dye only until the target intensity is reached, in contrast to regressive staining, which involves overstaining the tissue and then using acid alcohol to remove excess dye.

86. (D) Masson's Trichrome
Masson's Trichrome is a specialized histochemical stain widely used in diagnostic pathology to differentiate collagen from smooth muscle fibers. It relies on the sequential application of dyes of different molecular sizes, which selectively bind to specific tissue components, allowing the pathologist to easily evaluate fibrotic changes, organ scarring, and stromal reactions in chronic diseases.

87. (B) Blue or green
In tissue sections processed with Masson's Trichrome stain, mature collagen fibers are stained either deep blue or bright green, depending on whether aniline blue or light green is used as the counterstain. This distinct coloration provides a clear contrast against the red-stained muscle fibers and cytoplasm, and the dark brown-to-black stained cell nuclei.

88. (C) Picric acid and Acid Fuchsin
Van Gieson stain is a classic connective tissue stain consisting of a mixture of acid fuchsin and saturated picric acid. Acid fuchsin selectively stains collagen fibers a deep red, while picric acid serves as a counterstain, coloring cytoplasm, muscle, and erythrocytes yellow, providing a simple and effective method for demonstrating collagen.

89. (D) Apple-green birefringence
When tissue sections containing amyloid deposits are stained with Congo Red and examined under polarized light, they exhibit a pathognomonic apple-green birefringence. This optical phenomenon is due to the highly ordered, beta-pleated sheet molecular structure of amyloid, which binds the dye molecules in a parallel orientation, causing them to split polarized light.

110. (B) P-1, Q-2, R-3, S-4
Epoxy resins provide the exceptional hardness needed for ultrathin sectioning. The ultramicrotome is the specialized instrument designed to cut these sections at nanometer thickness. Lead citrate acts as a heavy metal stain that increases electron contrast on the sections. A diamond knife is the precision tool used within the ultramicrotome to cut specimens.

111. (C) 50 - 90 nanometers (nm)
Transmission electron microscopy utilizes an electron beam that has extremely low penetrating power through solid matter. To allow electrons to pass through the specimen and generate a high-contrast image, sections must be cut incredibly thin. The standard thickness range for these ultrathin sections is fifty to ninety nanometers, achieved using an ultramicrotome.

112. (C) Copper grids
Electrons cannot pass through standard glass microscope slides, which makes glass unsuitable for transmission electron microscopy. Instead, ultrathin tissue sections are mounted onto tiny metallic mesh grids, most commonly made of copper. These grids provide structural support to the delicate specimen while allowing the electron beam to pass freely through the open grid spaces.

113. (D) Lead citrate
Ultrathin sections of biological specimens lack natural contrast under an electron beam. To enhance visibility, heavy metal salts are applied as stains. Uranyl acetate primarily stains nucleic acids and proteins, while lead citrate acts as a counterstain that binds to proteins and lipid-associated complexes, significantly increasing overall image contrast in electron microscopy.

114. (C) The three-dimensional surface topography of a bulk specimen
Scanning electron microscopy functions by focusing an electron beam across the surface of a specimen. The interactions between the beam and the specimen surface release secondary electrons, which are detected to reconstruct a high-resolution, three-dimensional image. This imaging modality focuses specifically on surface topography and morphology rather than internal cellular structures.

115. (A) Both A and R are true and R is the correct explanation of A.
Preparing biological specimens for scanning electron microscopy requires removing all moisture. Evaporative drying creates severe surface tension at the liquid-gas interface, leading to the collapse of delicate surface structures. Critical point drying avoids this by transitioning the fluid under specific pressure and temperature where liquid and gas phases coexist, eliminating surface tension and preserving anatomy.

116. (D) To render non-conductive specimen surfaces electrically conductive
Biological tissues are naturally poor electrical conductors. When exposed to the high-energy electron beam in a scanning electron microscope, these specimens accumulate static charge, causing severe image distortion and thermal damage. Sputter coating deposits a micro-thin layer of gold-palladium on the specimen surface, rendering it electrically conductive and allowing charges to dissipate safely.

117. (A) Putrefaction
Early postmortem changes occur shortly after somatic death and include processes like rigor mortis, algor mortis, and livor mortis. These changes involve physical and chemical alterations of tissues without structural decay. Putrefaction represents a late postmortem change characterized by the bacterial decomposition and enzymatic breakdown of tissues, leading to gas production and green discoloration.

118. (C) The ambient environmental temperature and body insulation
Algor mortis is the gradual cooling of a carcass after death as it equilibrates with the environment. The rate of this cooling process is primarily governed by the temperature difference between the body and the ambient environment. Physical factors like body size, fat thickness, and insulating fur or fleece also strongly influence heat dissipation rates.

119. (A) Cadaveric spasm
Cadaveric spasm is an immediate postmortem stiffening of the musculature that bypasses the typical period of primary flaccidity. This condition occurs at the moment of death, usually associated with high-stress situations, extreme physical exhaustion, or severe central nervous system stimulation. It preserves the

Downloaded from www.pdf.tube

denatures most enzymes, rendering them inactive and impossible to detect histochemically.

101. (A) To break methylene bridges from formalin fixation and unmask epitopes
Formaldehyde preserves tissues by forming methylene bridges that cross-link proteins, which can mask antigenic epitopes and prevent antibody binding. Antigen retrieval is a crucial step in immunohistochemistry that uses heat or enzymes to break these cross-links, restoring the original three-dimensional structure of the epitopes and facilitating antibody access.

102. (D) Heat Induced Epitope Retrieval (HIER)
Heat Induced Epitope Retrieval is a standard technique in immunohistochemistry that exposes hidden antigenic sites. By heating formalin-fixed tissue sections in a buffer solution using a microwave, pressure cooker, or water bath, the cross-linked proteins are unfolded, restoring the conformation of the target epitopes and significantly enhancing antibody binding sensitivity.

103. (B) It quenches endogenous peroxidases to prevent non-specific background staining.
Many tissues contain active endogenous peroxidase enzymes, particularly red blood cells and granulocytes. Treating the section with hydrogen peroxide before applying the horseradish peroxidase-conjugated antibody inactivates these endogenous enzymes, preventing them from reacting with the chromogen and causing non-specific background staining that could easily obscure the true positive diagnostic signal.

104. (C) Brown
In immunohistochemistry, 3,3'-Diaminobenzidine is a widely used chromogen that serves as a substrate for horseradish peroxidase. In the presence of hydrogen peroxide, the enzyme catalyzes the oxidation of the chromogen, producing an insoluble, dark brown precipitate at the site of the antigen-antibody complex, which is easily visualized under light microscopy.

105. (C) A is true but R is false.
Direct immunofluorescence involves a single step where the primary antibody is directly conjugated to a fluorophore, which is faster but less sensitive. Indirect immunofluorescence, which uses an unlabeled primary antibody followed by a conjugated secondary antibody, is more sensitive because multiple secondary antibodies can bind to a single primary antibody, amplifying the signal.

106. (C) Applying an aqueous mounting medium to prevent quenching
Immunofluorescence microscopy relies on the detection of fluorescent signals from labeled antibodies within a tissue section. Organic mounting media, such as xylene-based formulations, can rapidly quench or dissolve these fluorophores. Utilizing an aqueous mounting medium prevents this rapid signal loss and maintains the excitation and emission capabilities of the fluorescent probe during examination.

107. (D) Glutaraldehyde
Transmission electron microscopy requires the preservation of fine cellular structures at the nanometer scale. Glutaraldehyde is a dialdehyde possessing two active aldehyde groups that rapidly cross-link proteins to stabilize cellular components. This rapid structural stabilization prevents autolysis and preserves the intricate detail of organelles far better than standard monoadhesives like neutral buffered formalin.

108. (A) Osmium tetroxide
Primary fixation with glutaraldehyde stabilizes proteins but does not adequately preserve membrane lipids. Osmium tetroxide is applied as a secondary fixative because it chemically reacts with unsaturated lipids, cross-linking and stabilizing them. Additionally, as a heavy metal, osmium introduces substantial electron density to lipid membranes, enhancing contrast during transmission electron microscopy imaging.

NODIA

109. (B) It has a very slow rate of penetration into tissues.
Glutaraldehyde cross-links proteins extremely rapidly at the tissue margin, which creates a physical barrier that restricts further diffusion of the fixative. This slow penetration rate necessitates the use of tiny tissue specimens, typically measuring one cubic millimeter or less. Proper penetration ensures uniform fixation and prevents autolytic degradation of deeper structures.

patent, leading to severe venous congestion, edema, and subsequent tissue death. This pathologic condition of simultaneous mechanical blockage and vascular compromise is termed an acute strangulating obstruction.

130. (C) Incomplete impregnation with paraffin wax
Paraffin wax embedding requires complete replacement of the clearing agent with molten wax. If the impregnation step is incomplete or insufficient time is allowed, xylene remains trapped in the center of the tissue block. This residual solvent prevents the wax from solidifying, resulting in a soft block center that retains a strong odor of xylene.

131. (B) Efficiently pull out the clearing agent from the tissue
The impregnation step in tissue processing replaces the clearing agent inside tissues with paraffin wax. Applying a vacuum during this phase reduces atmospheric pressure, which accelerates the volatilization and removal of clearing solvents like xylene. This facilitates rapid and thorough penetration of the molten paraffin wax into all spaces of the tissue block.

132. (D) Increase Hematoxylin staining time or replace exhausted dye
In hematoxylin and eosin staining, hematoxylin is the basic dye responsible for staining acidic nuclear chromatin dark blue. Faint nuclear staining paired with bright cytoplasmic eosinophilia indicates insufficient nuclear staining. This issue can be resolved in subsequent batches by extending the exposure time to the hematoxylin solution or by replacing the exhausted dye with fresh stain.

133. (A) Carbon (Anthracosis)
Pigments are colored substances classified as endogenous or exogenous based on their origin. Endogenous pigments, including melanin, hemosiderin, and lipofuscin, are synthesized within the body. Exogenous pigments originate from outside the body and enter via inhalation, ingestion, or injection. Carbon dust is a common inhaled exogenous pigment that accumulates in lung macrophages, causing anthracosis.

134. (C) Heart and liver
Lipofuscin is a golden-brown, insoluble lipid-protein pigment that accumulates within the lysosomes of cells undergoing progressive wear and tear. It represents a hallmark of cellular aging and is particularly prominent in permanent or stable cell types. Consequently, it is most commonly observed in the cardiomyocytes of the heart and the hepatocytes of the liver.

135. (A) Neutrophils
Suppurative or purulent inflammation is a localized response characterized by the accumulation of fluid, necrotic cellular debris, and dead or dying inflammatory cells. The hallmark cellular component of this exudate is the neutrophil, which migrates to the site of injury in response to chemotactic signals, typically indicating an underlying acute bacterial infection.

136. (D) Parasitic or allergic reaction
Eosinophils are specialized granulocytes containing cytotoxic major basic protein, which is highly effective against larval and adult parasites. These cells also migrate to tissue sites during immediate hypersensitivity reactions, mediated by mast cell degranulation. Therefore, finding numerous eosinophils in a tissue section indicates an underlying parasitic infestation or a severe local allergic reaction.

137. (B) Proliferative, scabby, pustular lesions of the lips and muzzle
Contagious ecthyma, caused by a parapoxvirus, is a highly infectious disease of sheep and goats. The disease presents with highly characteristic proliferative, pustular, and scabby lesions. These crusty lesions typically develop on the mucocutaneous junctions of the lips, muzzle, and nostrils, occasionally spreading to the feet or teats, serving as a pathognomonic diagnostic finding.

138. (C) Foot-and-Mouth Disease Virus
Foot-and-Mouth Disease is a highly contagious viral infection of cloven-hoofed animals. While adults develop painful vesicles, young calves can die acutely from severe viral myocarditis. This myocardial damage appears macroscopically as pale linear streaks of degeneration and necrosis alternating with normal red muscle fibers, creating the classic and highly diagnostic tiger heart appearance.

139. (D) Visceral and articular gout
Poultry excrete nitrogenous waste as uric acid rather than urea. When renal function

Click Here to Buy book on Amazon

exact physical posture of the animal at its time of death.

120. (D) The presence of surrounding tissue inflammation and vital cell reactions
Distinguishing true antemortem pathology from postmortem artifacts is vital in pathology. Postmortem changes, such as the passive diffusion of bile, cause tissue discoloration but lack host cellular responses. Antemortem lesions are characterized by active vascular and cellular reactions, including congestion, fibrin deposition, and the infiltration of inflammatory cells like neutrophils into the affected surrounding tissues.

121. (B) Explicit deviation is required to preserve specific fragile evidence
Standard necropsy protocols ensure systematic and complete examination of all organ systems. However, in vetero-legal and forensic cases, rigid adherence to these procedures may accidentally destroy or contaminate fragile trace evidence. Deviating from standard steps is justified only when specialized preservation of evidence, such as securing gunshot residue or delicate toxicological traces, is required.

122. (C) Bouin's fluid
Bouin's fluid is a popular picric acid-based fixative containing formaldehyde and glacial acetic acid. The picric acid component imparts a distinct yellow color to tissues, which must be washed out with alcohol during processing to ensure proper staining. It provides excellent preservation of delicate structures, glycogen reserves, and endocrine tissues for histopathology.

123. (D) Iodine treatment followed by sodium thiosulfate
Zenker's fluid contains mercuric chloride, which deposits a highly refractile, dark crystalline mercury pigment in tissue sections during fixation. To remove this artifact before staining, tissue sections must be treated with an iodine solution to convert the mercury into soluble mercuric iodide. Sodium thiosulfate is then applied to clear the iodine and the mercury complex.

124. (A) Fresh or chilled intact brain or brainstem tissues
Rabies virus is neurotropic and concentrates within specific areas of the central nervous system. Diagnosing this disease requires fresh or chilled brain tissue, specifically from the hippocampus, cerebellum, and brainstem. Fresh tissue is essential for the direct fluorescent antibody test, which is the gold standard diagnostic method, as formalin fixation can interfere with antigen-antibody binding.

125. (B) Highly Pathogenic Avian Influenza
Highly Pathogenic Avian Influenza is an extremely contagious viral disease affecting poultry with significant zoonotic potential and high mortality rates. Under Indian veterinary regulations, it is classified as a compulsory notifiable disease. Detecting or suspecting this infection requires immediate reporting to state and national animal health authorities to trigger swift containment measures.

126. (B) Bilirubin
Icterus, commonly known as jaundice, is a clinical and pathological state characterized by the yellowish discoloration of tissues, mucous membranes, and sclera. This condition arises from the pathological accumulation of bilirubin in blood and tissues. Bilirubin is a yellow pigment produced during the physiological and pathological breakdown of senescent red blood cells.

127. (A) Infarct
An infarct is a localized area of ischemic necrosis in an organ or tissue. It is caused by the sudden occlusion of either its arterial blood supply or its venous drainage. Common causes include thrombosis, embolism, or external compression of vessels. The resulting lack of oxygen and nutrients leads to rapid coagulative necrosis of parenchymal cells.

NODIA

128. (B) 1:3
During a cardiac necropsy, evaluating ventricular wall thickness is essential to diagnose hypertrophy or dilation. The left ventricle naturally has a thicker myocardium because it pumps blood against the high resistance of systemic circulation. In healthy dogs, the normal ratio of right ventricular thickness to left ventricular thickness is approximately one to three.

129. (D) Acute strangulating obstruction
Intestinal torsion in horses mechanically rotates the bowel along its mesenteric axis. This rotation compresses the thin-walled veins and lymphatic vessels while arterial inflow remains temporarily

exposed to unbuffered, acidic formalin solutions. The acid reacts with hemoglobin to form acid hematin, which deposits as dark brown, microcrystalline precipitates within and around blood vessels, potentially obscuring natural cellular details during microscopic evaluation of sections.

70. (B) Dehydration, Clearing, Impregnation, Embedding
Standard tissue processing is a sequential, multi-step procedure designed to prepare delicate specimens for paraffin embedding. The tissue is first dehydrated in ascending grades of alcohol to remove water, cleared using a solvent like xylene to replace the alcohol, impregnated with molten paraffin wax, and finally embedded to form a solid block.

71. (B) Replace tissue water with alcohol for subsequent hydrophobic media infiltration
The dehydration step is essential because paraffin wax, the standard embedding medium, is hydrophobic and cannot penetrate water-saturated tissues. By processing the tissue through ascending concentrations of alcohol, all free and bound water is gradually extracted and replaced, preparing the tissue for subsequent infiltration with clearing solvents and paraffin.

72. (C) Xylene
Xylene is the most widely used clearing agent in routine histology laboratories. It serves as an essential chemical intermediary because it is miscible with both the dehydrating alcohols and the hydrophobic paraffin wax, effectively removing the alcohol from the tissue and making the cells receptive to paraffin infiltration during embedding.

73. (D) It minimizes shrinkage and hardening of delicate tissues.
Unlike xylene, which can cause significant tissue hardening and excessive shrinkage if specimens are left exposed for too long, cedarwood oil is a gentle clearing agent. It clears tissues slowly and preserves their natural elasticity, making it ideal for processing delicate specimens such as brain, embryos, or fibrous tissues.

74. (C) 56-60°C
Histological grade paraffin wax is formulated to melt within this specific temperature range. Maintaining the wax at this temperature during tissue infiltration and embedding ensures optimal wax penetration without causing thermal damage, excessive shrinkage, or hardening of the delicate cellular structures, which would compromise subsequent sectioning and slide staining.

75. (C) II, I, III, IV
After embedding, the block is trimmed to expose the tissue and the knife angle is adjusted. The microtome is used to cut thin sections, which are then floated on a warm water bath to remove wrinkles. Finally, the flattened section is picked up onto a clean glass slide for drying and staining.

76. (B) Rotary microtome
The rotary microtome is the standard instrument used in diagnostic laboratories for cutting paraffin-embedded tissue blocks. It operates on a handwheel mechanism that converts rotary motion into vertical motion, allowing the operator to cut highly consistent, ultra-thin sections of tissue, typically ranging from three to five micrometers in thickness.

77. (B) -20°C to -30°C
This specialized microtome operates within a freezing cabinet maintained at this temperature range, which provides the optimal physical consistency for sectioning unfixed, frozen tissues. This technique allows for rapid diagnostic evaluation, particularly when preserving enzyme activity, lipids, or antigenicity that would be destroyed by routine paraffin processing.

78. (C) Ribbon formation
Ribbon formation occurs when consecutive paraffin sections adhere to one another along their edges during microtomy, creating a continuous chain of sections. This is highly desirable because it allows the laboratory technician to easily handle and float multiple sequential sections on a water bath, facilitating the selection of representative slices.

79. (D) There is a nick in the microtome knife edge.
Consistent vertical scratches, splits, or lines running through a tissue ribbon are typically caused by a damaged microtome blade. Even a minor nick on the cutting edge can slice through the paraffin block and tissue instead of cutting it cleanly, requiring the technician to shift the blade or replace it.

Downloaded from www.pdf.tube

is impaired, uric acid accumulates in the bloodstream and precipitates as white, chalky sodium urate crystals, known as tophi. Visceral gout is characterized by these white deposits coating the serosal surfaces of the pericardium and liver, while articular gout involves the joints.

140. (A) Marek's disease
Marek's disease is a lymphoproliferative viral disease of chickens caused by a herpesvirus. A classic pathological feature of the neuropathic form is the infiltration of peripheral nerves by neoplastic T-lymphocytes. This infiltration causes marked swelling and a grayish, translucent discoloration of major nerves, such as the sciatic and brachial nerves, with loss of their normal cross-striations.

141. (B) World Organisation for Animal Health (WOAH)
The World Organisation for Animal Health is the intergovernmental body responsible for improving animal health worldwide. This organization sets the global sanitary standards for international trade of animals and animal products. Veterinary health certificates issued for export must strictly comply with the guidelines and disease-free declarations established within their international terrestrial and aquatic codes.

142. (B) Decalcification
Bone tissue contains dense mineralized extracellular matrix containing calcium phosphate salts. This mineral density makes bone too hard to cut during routine microtomy, as it would damage the knife edge. Decalcification is the essential step performed after fixation to remove these calcium ions, softening the tissue so it can be easily sectioned and processed.

143. (A) Nitric acid
Decalcifying agents are used to remove calcium salts from bone. Strong mineral acids, particularly nitric acid, are highly effective and are commonly employed when rapid decalcification of dense cortical bone is required. While nitric acid operates very quickly, the duration must be carefully monitored to prevent the destruction of cellular detail and tissue staining characteristics.

144. (C) Saline prevents chemical reaction or leaching of toxic compounds
Standard formalin fixatives chemically cross-link proteins, which can degrade, react with, or mask toxic compounds in tissues, hindering forensic analysis. Preserving tissues in a saturated saline solution halts bacterial decomposition without chemically altering the specimen. This ensures that volatile poisons, heavy metals, and therapeutic drugs remain stable and extractable for accurate quantitative toxicological screening.

145. (D) An expert witness providing medical and pathological evidence
During a magisterial or police inquest in vetero-legal cases, the veterinarian performs the scientific postmortem examination to determine the cause and manner of death. In the subsequent legal proceedings, the veterinarian does not act as a prosecutor or investigator. Instead, they serve as an expert witness, providing objective, factual medical and pathological evidence to the court.

146. (C) Livor mortis
Livor mortis, also known as postmortem hypostasis, is the gravity-induced settling of blood within the capillaries and venules of the lowest portions of a carcass after circulation stops. This settling results in a distinct dark reddish-purple discoloration of the dependent skin and internal organs, helping pathologists determine the animal's physical orientation post-death.

147. (B) Interstitial pneumonia or emphysema
Normal lungs collapse immediately when the negative intrathoracic pressure is lost during chest cavity opening. Lungs that remain fully expanded and exhibit a rubbery, elastic texture are highly indicative of interstitial pneumonia or pulmonary emphysema. This texture reflects diffuse thickening of alveolar septa by inflammatory cells, edema, or fibrosis, which prevents normal elastic recoil.

148. (D) Fluorescent antibody testing on brain smears
A definitive postmortem diagnosis of rabies requires rapid and highly sensitive detection of viral antigens. While histopathological observation of Negri bodies in neurons can support a diagnosis, it is not completely reliable. The international gold standard is the direct fluorescent antibody test performed on fresh brain smears, which provides rapid, highly specific confirmation of the virus.

NODIA

90. (B) P-4, Q-1, R-3, S-2
Special stains are formulated to target specific biochemical components within tissues. The Periodic Acid-Schiff reaction demonstrates carbohydrates and glycogen, while Oil Red O is used to visualize neutral lipids. Alcian Blue targets acidic mucosubstances, and Congo Red is highly specific for demonstrating amyloid fibrils under both normal and polarized light microscopy.

91. (B) Magenta
A positive Periodic Acid-Schiff reaction is indicated by a striking, bright magenta or purplish-red color in the target structures. This distinct coloration occurs when periodic acid oxidizes adjacent glycol groups in carbohydrates to form aldehydes, which then react with Schiff's reagent to produce a stable, colored chromogenic complex in the tissue.

92. (B) Diastase (Amylase)
To confirm that a PAS-positive substance is glycogen, the tissue is treated with diastase or amylase before staining. Diastase enzymatically digests and removes glycogen, leaving other carbohydrates intact. If the magenta staining disappears in the enzyme-treated section compared to an untreated control, the substance is confirmed as glycogen.

93. (A) Sudan Black B for lipids
Lipids are highly soluble in the organic solvents, such as alcohols and xylene, used during routine paraffin tissue processing. Consequently, to demonstrate fat deposits in tissue sections, the pathologist must use frozen sections that have not been exposed to these solvents, staining them with lipid-soluble dyes like Sudan Black B.

94. (C) Mycolic acid
The Ziehl-Neelsen stain is designed to identify acid-fast organisms, such as Mycobacterium species. The cell walls of these bacteria contain high concentrations of mycolic acid, a waxy lipid that binds carbol fuchsin tightly and resists decolorization by acid-alcohol, allowing the red bacilli to stand out against a blue background.

95. (D) I, II and III
Histochemical stains are invaluable for identifying specific pigments and minerals in tissue sections. Perls' Prussian blue detects ferric iron, Von Kossa visualizes calcium deposits by replacing them with metallic silver, and Masson-Fontana demonstrates melanin based on its strong reducing properties, providing pathologists with critical diagnostic tools.

96. (C) Spirochetes
Warthin-Starry silver impregnation is the standard histochemical method for demonstrating spirochetes in tissue sections. Spirochetes are poorly visualized with routine H&E stains due to their thin, spiral morphology, but they can reduce silver ions from the stain solution to metallic silver, which deposits on their outer membranes, coloring them black.

97. (B) Grocott's Methenamine Silver (GMS)
Grocott's Methenamine Silver stain is highly regarded for demonstrating fungal organisms in tissue sections. Chromic acid is used to oxidize mucopolysaccharides in the fungal cell wall to aldehydes, which then reduce methenamine silver, depositing black metallic silver on the fungi and creating high contrast against a pale green counterstained background.

98. (D) Preserved as fresh frozen sections without harsh fixation or heat
Demonstrating enzyme activity in tissues requires preserving their native conformation and catalytic function, which are easily denatured by heat and chemical fixatives. Therefore, enzyme histochemistry is performed on fresh, unfixed, or minimally fixed frozen sections cut in a cryostat, avoiding the harsh dehydration, clearing, and embedding steps of routine histology.

99. (A) Azo dye
In simultaneous coupling methods, the target enzyme hydrolyzes a synthetic substrate to release a primary reaction product. This product immediately reacts with a diazonium salt present in the incubation medium to form an insoluble, highly colored azo dye precipitate at the exact site of enzyme activity, allowing precise microscopic localization of the enzyme.

100. (B) II and III
Dehydrogenases are demonstrated using tetrazolium salts as electron acceptors, while esterase stains help identify specific inflammatory cell lineages. In contrast, alkaline phosphatase operates optimally in basic conditions, and heat must be avoided during processing because it

Click Here to Buy book on Flipkart

a defined zone. This action is followed by the complete destruction of carcasses, disinfection of premises, and temporary quarantines to stop viral transmission.

159. (A) Loss of the delicate epithelial lining and villi
The mucosal lining of the gastrointestinal tract is highly fragile and prone to rapid autolysis. During necropsy, scraping or rough handling of the mucosal surface can mechanically strip away the delicate single-cell layer of epithelial cells and villous tips. Avoiding this trauma is critical to preserving mucosal architecture for accurate histopathological assessment under the microscope.

160. (C) Fibrinous pericarditis
Fibrinous pericarditis is characterized by the accumulation of a thick exudate containing fibrin on the epicardial and pericardial surfaces. When these two adhering membranes are separated during postmortem examination, the rough, shaggy yellow-gray surface resembles buttered bread that has been pulled apart. This distinct gross appearance is classic for acute fibrinous inflammation.

161. (D) Wedge-shaped, pale area with its base at the capsule
The kidney and spleen possess a highly structured, terminal arborizing arterial blood supply with minimal collateral circulation. An arterial occlusion in these organs results in a classic wedge-shaped area of ischemic coagulative necrosis. The wide base of this wedge-shaped pale infarct aligns with the outer organ capsule, while the apex points toward the site of vascular occlusion.

162. (C) Thymus
The thymus is a primary lymphoid organ that is highly developed in young, unweaned animals and plays a critical role in T-lymphocyte maturation. It is highly sensitive to physiological stress, nutritional deprivation, and viral infections, which trigger rapid atrophy. Evaluating the thymus first during a thoracic necropsy provides vital information regarding the animal's baseline immune status.

163. (B) Prevent leakage of ingesta and contamination
Ruminant digestive tracts contain large volumes of fluid, partially digested ingesta, and abundant microflora. During postmortem extraction of the visceral organs, there is a high risk of spilling this material. Double ligating the esophagus and rectum before removal prevents leakage of these contents, protecting the thoracic and abdominal cavities from severe contamination during the necropsy.

164. (D) Immunohistochemistry or ELISA on the brainstem obex
Bovine spongiform encephalopathy is a fatal neurodegenerative disease caused by abnormal prion proteins. These infectious prions selectively accumulate in specific regions of the central nervous system. The official postmortem diagnostic protocol requires collecting the obex region of the brainstem. Confirming the disease is achieved by demonstrating the prion protein using immunohistochemistry or rapid enzyme-linked immunosorbent assays.

165. (A) The tissue block face is compressed and sections skip
During microtomy, the clearance angle is the angle between the cutting facet of the knife and the block face. If this angle is adjusted too small, the lower facet of the blade rubs flatly against the tissue block. This physical contact compresses the block face rather than shearing it, resulting in failed cuts or skipped sections.

166. (C) Very large or hard tissue blocks like whole brain or bone
The sledge microtome is a heavy-duty sectioning instrument where either the knife or the specimen block slides along a heavy horizontal track. This design provides maximum stability and cutting force. It is utilized primarily for sectioning exceptionally large or hard tissue blocks, such as whole organs, large brain slices, or dense decalcified bone specimens.

167. (A) Rabies
Negri bodies are round or oval, sharp-bordered, eosinophilic, intracytoplasmic inclusion bodies. They represent accumulations of viral proteins and nucleocapsids within infected neurons. Finding these inclusion bodies, particularly within the pyramidal cells of the hippocampus or the Purkinje cells of the cerebellum, is highly diagnostic and pathognomonic for a rabies virus infection.

Downloaded from www.pdf.tube

(A) Hypoproteinemia
(B) Presence of target cells
(C) Severe thrombocytopenia
(D) Lipemia

2004

10. Assess the validity of the following assertion and reason regarding serum analysis and choose the correct option:
Assertion (A): Hemolysis in a serum sample often leads to artificially elevated levels of lactate dehydrogenase (LDH).
Reason (R): Erythocytes contain high concentrations of LDH, which is released into the serum upon cell membrane rupture.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2005

11. Which artifact is most commonly associated with excessive pressure applied during the preparation of a fine needle aspiration smear?
(A) Formation of rouleaux and erythrocyte clumping
(B) Nuclear streaming and cellular distortion
(C) Intense background staining with basophilic debris
(D) Spontaneous cellular lysis and phagocytosis

2003

12. During erythrogram evaluation, how is the Mean Corpuscular Hemoglobin Concentration (MCHC) calculated?
(A) $\frac{PCV}{\text{Hemoglobin}} \times 10$
(B) $\frac{\text{Hemoglobin}}{PCV} \times 100$
(C) $\frac{\text{Hemoglobin}}{TEC} \times 10$
(D) $\frac{TEC}{PCV} \times 100$

2001

13. Which condition is classically indicated by an increased Mean Corpuscular Volume (MCV) and a decreased Mean Corpuscular Hemoglobin Concentration (MCHC)?
(A) Regenerative anemia
(B) Iron deficiency anemia
(C) Anemia of chronic disease
(D) Aplastic anemia

2015

14. What does the term "anisocytosis" specifically refer to in the morphological evaluation of erythrocytes?
(A) Variation in cell shape
(B) Presence of abnormal cellular inclusions
(C) Variation in cell size
(D) Altered hemoglobin distribution within the cell

2011

15. The presence of spherocytes on a canine blood smear is highly suggestive of which underlying pathophysiological mechanism?
(A) Oxidative damage to hemoglobin
(B) Iron deficiency leading to altered membrane structure
(C) Bone marrow hypoplasia
(D) Immune-mediated destruction of erythrocytes

2013

16. Which inclusion body is formed by the denaturation and precipitation of hemoglobin due to oxidative stress, such as onion toxicity in dogs?
(A) Howell-Jolly bodies
(B) Basophilic stippling
(C) Heinz bodies
(D) Pappenheimer bodies

2015

17. A left shift in a leukogram is best defined by the increased presence of which specific cell type in peripheral circulation?
(A) Hypersegmented and mature neutrophils
(B) Band and immature neutrophils
(C) Reactive and atypical lymphocytes
(D) Activated tissue-derived macrophages

2002

18. What is the most appropriate diagnostic interpretation of a "degenerative left shift" in a complete blood count?
(A) Mature neutrophils outnumber immature forms with a marked, persistent leukocytosis.
(B) The total leukocyte count remains elevated, consisting of reactive lymphocytes.
(C) Hypersegmented neutrophils predominate because of a prolonged circulatory half-life.
(D) Immature neutrophils outnumber mature neutrophils, usually with leukopenia.

NODIA

and subacute, is highly characteristic of a viral infection within the central nervous system.

178. (C) Watertight, leak-proof, and wrapped in absorbent material
Transporting liquid biological specimens safely requires adhering to strict biosafety standards. The primary receptacle, which directly contains the liquid sample, must be completely watertight and leak-proof to prevent escape of pathogens. It must also be wrapped in sufficient absorbent material to capture the entire fluid volume in the event of physical breakage or leakage.

179. (B) Electromagnetic lenses
Electron microscopes utilize a high-energy electron beam instead of light waves to image specimens at nanometer resolution. Because electrons are charged particles, standard glass optical lenses cannot refract or focus them. Instead, electromagnetic lenses are arranged along the microscope column to focus and steer the electron beam onto the specimen and focus the final image.

180. (D) Pantothenic acid
Pantothenic acid is an essential B-vitamin required for coenzyme A synthesis. In growing swine, a deficiency of this nutrient leads to progressive peripheral nerve damage. Histologically, this presents as Wallerian degeneration of sensory and motor axons. Clinically, this neuropathy manifests as a characteristic, stiff-legged walking style known as a goose-stepping gait.

181. (A) Severe fibrinous epicarditis
A cor villosum, commonly referred to as a shaggy heart, is a classic gross pathological description. This appearance occurs during severe fibrinous epicarditis when a thick layer of fibrin deposits on the visceral pericardium. The continuous movement of the beating heart stretches and pulls this sticky fibrin into rough, hair-like projections across the cardiac surface.

182. (C) Frozen sectioning using a cryostat
Standard paraffin processing of tissue biopsies requires several hours or days to complete. For rapid intraoperative diagnosis while a patient is on the operating table, frozen sectioning is utilized. This technique involves freezing fresh tissue instantly and cutting sections using a microtome housed in a cold chamber, known as a cryostat, providing results within minutes.

183. (B) Masson's trichrome
Masson's trichrome is a classic multi-step histological stain used to differentiate collagen from other tissue elements. Under this staining protocol, collagen fibers are stained brilliant blue or aniline green, while muscle fibers, cytoplasm, and keratin stain red. This contrast makes it highly valuable for evaluating fibrosis, cirrhosis, and connective tissue tumors in histopathology.

184. (D) Softening or liquefaction of tissue
The suffix malacia is derived from the Greek word for softness and is used in pathology to denote the abnormal softening of a tissue or organ. This softening is typically the result of ischemic necrosis, enzymatic degradation, or liquefactive necrosis. Examples include encephalomalacia in the brain and osteomalacia in bone tissue.

185. (B) Volvulus
Volvulus is a specific form of mechanical bowel obstruction characterized by the twisting of a segment of intestine around its mesenteric axis. This rotation compromises the mesenteric blood vessels, leading to rapid venous congestion and hemorrhagic infarction. In equine medicine, volvulus of the small intestine is a critical, life-threatening cause of acute colic.

186. (C) Potassium alum
Hematoxylin is a natural dye that requires a metallic mordant to bind to tissue components. In the formulation of Harris's hematoxylin, potassium aluminum sulfate, commonly known as potassium alum, is utilized as the mordant. This metal-dye complex binds strongly to the acidic phosphate groups of nuclear DNA, resulting in the characteristic blue staining.

187. (D) Incomplete lung expansion or collapse
Atelectasis is a pathological term describing the incomplete expansion of alveoli at birth or the collapse of previously inflated lung parenchyma. Grossly, atelectatic lungs appear dark red, sunken, and fleshy or liver-like, and they fail to float in water. This condition reduces the available surface area for gas exchange, causing hypoxia.

188. (C) Fowl Pox
Fowl pox is a slow-spreading viral disease of poultry caused by an avipoxvirus. The cutaneous or dry form of this disease is characterized by the development of proliferative, nodular, and crusty

Click Here to Buy book on Amazon

168. (D) Macule
A macule is a circumscribed, flat area of skin or mucosal discoloration. It is distinguished by being level with the surrounding tissue, meaning it is non-palpable and presents no elevation or depression. By definition, a macule measures less than one centimeter in diameter, representing a localized change in pigment without structural thickening.

169. (B) Abrasions are superficial friction scrapes; lacerations are irregular blunt force tears
Abrasions and lacerations are distinct mechanical injuries encountered in veterinary forensics. An abrasion is a superficial injury involving the scraping or wearing away of the epidermis by friction against a rough surface. A laceration is a deeper, irregular tear in the skin and underlying tissues caused by blunt force impact, typically featuring tissue bridging.

170. (C) Amyl acetate
Critical point drying requires a fluid that is miscible with both the dehydration agent and liquid carbon dioxide. Amyl acetate is commonly employed as this transitional fluid. It completely replaces absolute ethanol in the tissue and is subsequently easily replaced by liquid carbon dioxide within the drying chamber, facilitating smooth transition through the critical point.

171. (D) It binds to the primary antibody and carries the enzyme label
Immunohistochemistry utilizes a multi-step antibody system to detect target antigens. The primary antibody binds directly and specifically to the tissue antigen of interest. The secondary antibody is designed to recognize and bind to the constant region of the primary antibody. This secondary antibody is conjugated to enzymes or biotin, amplifying the signal for visualization.

172. (A) Mucus
Catarrhal inflammation is a localized inflammatory response restricted to mucous membranes, particularly in the respiratory and gastrointestinal tracts. It is characterized by marked hypersecretion of mucus from goblet cells and mucosal glands, mixed with desquamated epithelial cells and inflammatory cells. This creates a thick, translucent, or cloudy fluid coating the affected tissue surface.

NODIA

173. (B) Anasarca
Anasarca is a severe and generalized form of edema. It is characterized by widespread accumulation of fluid within the subcutaneous tissues throughout the entire body, leading to diffuse swelling. This condition is often accompanied by fluid accumulation in major body cavities and typically results from severe hypoproteinemia, congestive heart failure, or renal dysfunction.

174. (B) Transmissible spongiform encephalopathy
Transmissible spongiform encephalopathies are fatal neurodegenerative diseases caused by prions. Under light microscopy, the hallmark histopathological lesion is spongiform change, characterized by the progressive development of clear, round vacuoles within the neuropil and neuronal cytoplasm. This microvacuolation creates a classic spongy or Swiss cheese appearance in the affected brain tissue.

175. (A) Ultramicrotome
The ultramicrotome is a highly specialized sectioning instrument designed for transmission electron microscopy preparation. It operates with extreme precision, utilizing thermal or mechanical advance mechanisms to feed the specimen in nanometer increments. The instrument cuts ultrathin sections using custom-prepared glass knives or high-grade diamond knives to achieve the required sub-micrometer thickness.

176. (C) Opening major body cavities and immersing the carcass in 10% NBF
Fixation of an entire small rodent carcass requires immediate access of the fixative to internal organs. Simply immersing an intact carcass fails because the skin and abdominal wall act as barriers, leading to rapid internal autolysis. Opening the thoracic and abdominal cavities before immersion ensures that neutral buffered formalin rapidly reaches and preserves all internal tissues.

177. (D) Viral
Encephalitis is classified based on the character of the inflammatory exudate. Non-suppurative inflammation is characterized by the absence of neutrophils and the predominance of lymphocytes, plasma cells, and macrophages, often accompanied by perivascular cuffing. This histological pattern, especially when diffuse

198. (C) The animal is being transported across borders
Veterinary health certificates are legal documents issued by authorized veterinarians. The primary indication for issuing this certificate is when animals are being transported across state, national, or international borders. The document officially verifies that the animals have been examined, are free from clinical signs of infectious disease, and meet the import regulations of the destination.

199. (A) Serous atrophy of fat in the coronary groove and bone marrow
Starvation forces the body to mobilize its remaining adipose reserves for energy. This process results in serous atrophy of fat, also known as gelatinous degeneration. Grossly, the fat deposits in the coronary groove of the heart, perirenal space, and bone marrow lose their firm white texture, becoming translucent, yellow, and gelatinous. This finding confirms severe cachexia.

200. (D) Normal tissue components within the patient's section that naturally express the target antigen
In immunohistochemistry, an internal positive control refers to normal, non-neoplastic tissue components present within the patient's own slide that naturally express the target antigen. Observing positive staining in these normal cells validates the entire staining run. It proves that the primary antibody, secondary antibody, and detection system are functioning correctly without needing a separate control slide.

201. (B) Acute exudation of fibrinogen forming fibrin networks
The term fibrinous describes a specific acute inflammatory exudate. This occurs when severe vascular injury allows high-molecular-weight plasma proteins, specifically fibrinogen, to leak out of blood vessels into surrounding tissues or body cavities. Once extravascular, fibrinogen polymerizes into an insoluble meshwork of fibrin, appearing microscopically as an eosinophilic network of delicate threads.

202. (C) The pelvic flexure of the large colon
The equine large colon is anatomically complex, featuring sudden changes in luminal diameter and direction. The pelvic flexure is a highly critical region where the wide left ventral colon transitions into the narrow left dorsal colon while making a sharp turn. This abrupt narrowing makes the pelvic flexure the most common site for gastrointestinal impaction and colic.

203. (D) Collecting samples to confirm strain for containment
Highly Pathogenic Avian Influenza is a devastating disease of poultry. During an outbreak, the primary responsibility of a veterinary pathologist performing field necropsies is biosafety and disease surveillance. The pathologist must systematically collect and package appropriate tissue samples, such as trachea and brain, to confirm viral strain and subtype, which guides quarantine and containment efforts.

204. (A) Johne's disease (Paratuberculosis)
Johne's disease is a chronic wasting infection of ruminants caused by Mycobacterium avium subspecies paratuberculosis. The bacterium targets the distal small intestine, triggering a diffuse granulomatous inflammatory response. This chronic macrophage infiltration causes massive, sausage-like thickening of the intestinal mucosa with prominent, permanent transverse corrugations. This classic mucosal thickening is pathognomonic for the disease.

205. (B) Trichobezoar
Bezoars are tightly compacted masses of indigestible material that accumulate within the gastrointestinal tract, most commonly in the stomach or rumen. When these masses are composed entirely of hair, usually ingested during grooming or licking due to mineral deficiencies, they are termed trichobezoars. Phytobezoars, by contrast, are composed of plant fibers.

206. (B) Carbon monoxide or cyanide poisoning
Carbon monoxide and cyanide poisonings interfere with normal cellular respiration. Carbon monoxide binds to hemoglobin forming carboxyhemoglobin, which has a distinct bright red color. Cyanide inhibits cytochrome c oxidase, preventing tissues from utilizing oxygen, which leaves venous blood highly oxygenated. Both conditions result in a characteristic bright cherry-red coloration of blood and tissues postmortem.

207. (A) Chocolate-brown colored blood
Nitrate poisoning occurs when ruminants ingest plants high in nitrates, which are reduced to nitrites in the rumen. Nitrites oxidize the ferrous iron in hemoglobin to ferric iron, forming

Downloaded from www.pdf.tube

lesions. These lesions are typically restricted to the unfeathered skin of the bird, such as the comb, wattles, eyelids, head, and feet.

189. (B) Classical Swine Fever
Classical Swine Fever is a highly contagious viral disease of pigs. The virus causes severe systemic vasculitis and endothelial damage, leading to widespread hemorrhages. Macroscopically, this damage is highly visible on the renal cortex as multiple pinpoint petechial hemorrhages. This distinctive presentation is classically referred to as a flea-bitten kidney during postmortem examination.

190. (B) The fluorochrome absorbs shorter wavelengths and emits longer wavelengths
Fluorochromes are specialized molecules that exhibit luminescence when excited by electromagnetic radiation. The fundamental principle governing this emission is Stokes shift. When a fluorochrome is hit by light of a specific shorter wavelength, it absorbs this high-energy light. It then rapidly emits light at a longer, lower-energy wavelength, which is detected by the microscope.

191. (A) It dictates the plane of sectioning to keep key layers visible
Embedding is the process of enclosing tissues in a support medium like paraffin wax. Ensuring correct orientation of the tissue specimen within the mold is crucial. This step dictates the exact plane of sectioning during microtomy. Proper orientation ensures that all vital anatomical layers, such as the mucosa and muscularis of intestines, are visible on the slide.

192. (C) Postmortem autolysis by gastric acids
Following death, the mucosal barriers of the digestive tract fail, allowing active digestive juices and gastric acids to degrade the tissue. This rapid postmortem autolysis causes mucosal peeling, thinning, and dark discoloration of the stomach or rumen lining. This autolytic breakdown can easily mimic true antemortem ulceration or necrosis if the necropsy is delayed.

193. (D) Hemosiderin-laden alveolar macrophages
Left-sided heart failure leads to chronic pulmonary congestion and increased hydrostatic pressure in capillaries. This pressure forces red blood cells into the alveoli, where they are phagocytized by resident alveolar macrophages. These macrophages degrade hemoglobin into brown hemosiderin pigment. The presence of these hemosiderin-laden macrophages in alveolar spaces is diagnostic for heart failure.

194. (C) Between the secondary and outer packaging in a waterproof pouch
The triple packaging system maintains biosafety and prevents specimen contamination. While the sample is secured in the primary and secondary layers, accompanying paperwork must remain dry and readable. Therefore, submission forms and diagnostic requests should be placed in a waterproof pouch located between the secondary packaging and the outer rigid shipping container to protect them from leaks.

195. (B) Perenyi's fluid
Perenyi's fluid is a specialized chemical formulation containing nitric acid, absolute alcohol, and chromic acid. This unique mixture performs two functions simultaneously by fixing tissue proteins while extracting calcium salts from mineralized structures. It is highly valued because it decalcifies tissues gently and avoids the severe swelling or tissue distortion caused by stronger unbuffered decalcifiers.

NODIA

196. (D) Soft, pliable bones in adults due to deficient osteoid mineralization
Osteomalacia is a metabolic bone disease of mature adult animals. It is characterized by a failure of the osteoid matrix to undergo proper mineralization, typically due to phosphorus or vitamin D deficiency. This defect leaves the bone structure dominated by uncalcified osteoid, rendering the skeleton soft, pliable, and prone to pathological fractures under normal weight-bearing.

197. (A) Bovine Tuberculosis
Bovine Tuberculosis, caused by Mycobacterium bovis, is a chronic infectious disease. The pathognomonic lesion is the tubercle, which is a granuloma characterized by central caseous necrosis and calcification. These lesions are surrounded by epithelioid macrophages, lymphocytes, and multinucleated giant cells, presenting grossly as firm, yellowish nodules within the lungs and regional lymph nodes.

CHAPTER 08
Clinical Pathology

2024

1. Which of the following best defines the primary purpose of adding a preservative like boric acid to a urine sample during transport?
(A) To prevent the precipitation of struvite crystals
(B) To inhibit bacterial overgrowth while preserving cellular elements
(C) To maintain the natural color and transparency of the sample
(D) To solubilize amorphous urates and phosphates

2007

2. For evaluating blood glucose levels in a sample delayed in transit, which collection tube is most appropriate?
(A) K3-EDTA tube
(B) Plain red-top tube
(C) Sodium citrate tube
(D) Sodium fluoride tube

2014

3. What is a common artifact observed in a whole blood sample stored at room temperature for an extended period before making a smear?
(A) Echinocytosis and leukocyte pyknosis
(B) Basophilic stippling of erythrocytes
(C) Spontaneous formation of Heinz bodies
(D) Microcytosis due to cellular shrinkage

2004

4. Which specific pre-analytical error is most likely to artificially elevate serum potassium levels in a canine sample?
(A) Fasting the patient for 12 hours prior to collection
(B) Using a large-bore needle for venipuncture
(C) Delayed separation of serum from erythrocytes
(D) Immediate centrifugation of the blood sample

2011

5. The mechanism of action of EDTA as an anticoagulant involves which of the following processes?
(A) Chelation of ionized calcium
(B) Activation of antithrombin III
(C) Inhibition of prothrombin synthesis
(D) Binding of tissue factor

2023

6. Why is sodium citrate the preferred anticoagulant for coagulation profiling (e.g., PT and aPTT) rather than EDTA?
(A) It irreversibly chelates calcium, completely halting the coagulation cascade
(B) It prevents platelet clumping more effectively than EDTA
(C) It stabilizes factors V and VIII better than any other anticoagulant
(D) Its calcium-binding effect is easily reversible by adding calcium chloride

2014

7. Based on its mechanism of action, which anticoagulant is widely considered the best choice for avian and reptilian hematology to avoid excessive cell lysis?
(A) Sodium citrate
(B) Lithium heparin
(C) K3-EDTA
(D) Sodium fluoride

2024

8. Identify the INCORRECT statement regarding sample handling and transport for veterinary clinical pathology.
(A) Whole blood for a complete blood count (CBC) should not be frozen.
(B) Serum should be separated from the clot within one hour to prevent artifactual hypoglycemia.
(C) Cytology slides should be shipped in the same sealed container as formalin-fixed tissues.
(D) Blood smears should be made immediately after collection to minimize morphological artifacts.

2004

9. In the context of pre-analytical errors, which factor directly causes a false increase in the spectrophotometric measurement of hemoglobin?

Click Here to Buy book on Flipkart

methemoglobin. Methemoglobin is incapable of transporting oxygen, causing systemic hypoxia. This high concentration of methemoglobin imparts a distinct chocolate-brown color to the blood and tissues.

208. (C) An entry wound
Gunshot wounds exhibit distinct pathological characteristics that help determine the trajectory of a projectile. An entry wound is typically characterized by an abrasion collar along its margins, where the bullet scrapes the skin upon contact. In flat bones or tissues, the force of entry pushes material inward, creating characteristic inward-beveled margins around the defect.

209. (D) It must be frozen unfixed or formalin-fixed for cryostat sectioning
Routine histopathological processing utilizes organic solvents like xylene and alcohols, which completely dissolve lipids, leaving behind empty vacuoles in tissue sections. To diagnose lipid storage diseases, tissue lipids must be preserved. This requires utilizing frozen, unfixed, or formalin-fixed tissue sectioned on a cryostat. This technique permits subsequent staining with lipid-soluble dyes like Oil Red O.

210. (C) Composition and atomic number differences
Scanning electron microscopes detect various signals resulting from electron-specimen interactions. Secondary electrons provide highly detailed information about surface topography. Backscattered electrons, however, are high-energy electrons reflected back by elastic collisions with atomic nuclei. The signal intensity depends on the average atomic number of the target area, providing contrast based on composition and material differences.

****

NODIA

2006

19. Physiological leukocytosis, commonly observed in excited or fearful young cats, is primarily mediated by which endogenous substance?
(A) Epinephrine
(B) Glucocorticoids
(C) Histamine
(D) Bradykinin

2003

20. Match the morphological abnormality in List I with its corresponding clinical indication in List II:
List I: (Morphological Abnormality)
P. Acanthocytes
Q. Schistocytes
R. Target cells (Codocytes)
S. Echinocytes
List II: (Clinical Indication)
1. Disseminated intravascular coagulation (DIC)
2. Hepatic lipidosis or hemangiosarcoma
3. Uremia or artifactual prolonged storage
4. Regenerative anemia or liver disease
(A) P-2, Q-1, R-4, S-3
(B) P-1, Q-2, R-3, S-4
(C) P-3, Q-4, R-1, S-2
(D) P-2, Q-4, R-1, S-3

2025

21. Which method is considered the most accurate manual technique for estimating the absolute thrombocyte count in veterinary laboratories?
(A) Hemocytometer counting using 1% ammonium oxalate diluent
(B) Examination of the buffy coat using a microhematocrit tube
(C) Estimation exclusively from the feather edge of a stained blood smear
(D) Calculation derived directly from the packed cell volume

2016

22. In the evaluation of coagulation disorders, an isolated prolongation of the activated partial thromboplastin time (aPTT) with a normal prothrombin time (PT) suggests a defect in which pathway?
(A) Extrinsic pathway
(B) Common pathway
(C) Fibrinolytic pathway
(D) Intrinsic pathway

NODIA

2002

23. What morphological feature distinguishes primary platelet clumping from true thrombocytopenia on a peripheral blood smear?
(A) Giant platelets distributed uniformly throughout the blood monolayer
(B) Large platelet aggregates at the feathered edge of the smear
(C) A complete absence of megakaryocytes in concurrent bone marrow
(D) Prominent toxic granulation present in the adjacent neutrophils

2002

24. Which intracellular hemoparasite of cattle is typically identified by pairs of piriform organisms forming an acute angle within the erythrocyte?
(A) Theileria annulata
(B) Anaplasma marginale
(C) Babesia bigemina
(D) Trypanosoma evansi

2006

25. Ehrlichia canis primarily infects which specific circulating cell type in dogs, leading to the formation of characteristic morulae?
(A) Erythrocytes
(B) Eosinophils
(C) Thrombocytes
(D) Monocytes

2004

26. Which liver enzyme is considered highly liver-specific in dogs and cats and primarily indicates hepatocellular leakage?
(A) Alanine Aminotransferase (ALT)
(B) Alkaline Phosphatase (ALP)
(C) Gamma-Glutamyl Transferase (GGT)
(D) Aspartate Aminotransferase (AST)

2009

27. In large animals such as horses and cattle, which enzyme is most reliably utilized as an indicator of hepatocellular damage due to the lack of significant diagnostic ALT activity?
(A) Alkaline Phosphatase (ALP)
(B) Sorbitol Dehydrogenase (SDH)
(C) Creatine Kinase (CK)
(D) Amylase

2010

28. An elevation in Alkaline Phosphatase (ALP) and Gamma-Glutamyl Transferase (GGT) without significant elevation in ALT is most characteristic of which pathological process?
(A) Acute hepatocellular necrosis

(B) Cholestasis
(C) Portosystemic shunting
(D) Hemolytic anemia

2014

29. Which biochemical parameter provides the most accurate reflection of hepatic synthetic function rather than cellular injury?
(A) Serum albumin
(B) Serum Alanine Aminotransferase (ALT)
(C) Serum Alkaline Phosphatase (ALP)
(D) Gamma-Glutamyl Transferase (GGT)

2014

30. Consider the following statements regarding bile acid testing in veterinary medicine:
I. Pre- and post-prandial bile acids are used to evaluate hepatic function.
II. Bile acids are synthesized in the liver from cholesterol.
III. Elevated bile acids are diagnostic specifically for cholangiocarcinoma.
Which of the above statements are correct?
(A) I and II
(B) II and III
(C) I only
(D) I, II and III

2013

31. Which scenario typically causes an elevation in unconjugated (indirect) bilirubin without a corresponding increase in conjugated (direct) bilirubin?
(A) Extrahepatic biliary duct obstruction
(B) Intrahepatic cholestasis due to lipidosis
(C) Chronic progressive renal failure
(D) Extravascular hemolytic anemia

2005

32. Symmetric dimethylarginine (SDMA) is utilized in veterinary medicine primarily as an early biomarker for assessing the function of which organ?
(A) Pancreas
(B) Liver
(C) Kidney
(D) Myocardium

2014

33. Why is serum creatinine generally considered a more specific indicator of glomerular filtration rate (GFR) than blood urea nitrogen (BUN)?
(A) Creatinine is synthesized entirely within the proximal renal tubules.
(B) BUN is influenced by dietary protein and gastrointestinal bleeding.
(C) Creatinine undergoes substantial tubular reabsorption under normal GFR.
(D) BUN requires active transport across the glomerular membrane barrier.

2019

34. In avian and reptilian clinical biochemistry, which metabolite replaces BUN as the primary indicator of renal function due to their uricotelic nitrogen metabolism?
(A) Creatinine
(B) Ammonia
(C) Allantoin
(D) Uric acid

2005

35. The finding of pre-renal azotemia is best differentiated from primary renal azotemia by evaluating which concurrent diagnostic parameter?
(A) Urine specific gravity
(B) Serum phosphorus concentration
(C) Serum calcium concentration
(D) Absolute reticulocyte count

2003

36. Which diagnostic test is considered the gold standard for diagnosing exocrine pancreatic insufficiency (EPI) in dogs?
(A) Serum amylase
(B) Serum lipase
(C) Trypsin-like immunoreactivity (TLI)
(D) Pancreatic lipase immunoreactivity (PLI)

2016

37. In evaluating gastrointestinal disease, low serum cobalamin combined with high serum folate is most strongly indicative of which condition?
(A) Small intestinal bacterial overgrowth (SIBO)
(B) Moderate exocrine pancreatic insufficiency (EPI)
(C) Severe gastric ulceration with mucosal bleeding
(D) Chronic proximal duodenal malabsorption syndrome

2012

38. Which cardiac biomarker is widely measured to diagnose myocardial injury in animals due to its high specificity for cardiac muscle damage?
(A) Creatine Kinase-MM (CK-MM)
(B) Aspartate Aminotransferase (AST)
(C) Alkaline Phosphatase (ALP)
(D) Cardiac Troponin I (cTnI)

Downloaded from www.pdf.tube

2023

39. To differentiate whether an elevated AST is of hepatic or skeletal muscle origin in a horse, which enzyme should be concurrently evaluated?
(A) GGT
(B) Creatine Kinase (CK)
(C) SDH
(D) Amylase

2009

40. Consider the following statements regarding serum proteins:
I. Albumin represents the largest single protein fraction in normal serum.
II. Fibrinogen is included in the measurement of total protein when serum is used instead of plasma.
III. Acute phase proteins like C-reactive protein (CRP) decrease significantly during acute inflammation.
IV. Globulin levels are typically calculated by subtracting albumin from total protein.
Which of the above statements are INCORRECT?
(A) I and IV
(B) II, III and IV
(C) II and III
(D) I, II and IV

2005

41. A decreased albumin-to-globulin (A:G) ratio due to an absolute hyperglobulinemia is most commonly associated with which pathological state?
(A) Protein-losing enteropathy
(B) Severe hepatic cirrhosis
(C) Acute blood loss
(D) Chronic antigenic stimulation

2007

42. In veterinary acid-base analysis, what is the primary expected compensatory mechanism for a primary metabolic acidosis?
(A) Hyperventilation resulting in a decreased PCO_2
(B) Decreased renal reabsorption of bicarbonate
(C) Hypoventilation resulting in an increased PCO_2
(D) Increased renal excretion of ammonium

2004

43. Which artifact is known to falsely decrease measured serum calcium levels in a biochemical profile?
(A) Prolonged venous stasis during collection
(B) Collection in an EDTA tube
(C) Hyperalbuminemia
(D) Use of a red-top serum tube

2013

44. What is the predominant intracellular cation that plays a critical role in maintaining resting membrane potential and is evaluated in serum electrolyte panels?
(A) Sodium
(B) Potassium
(C) Calcium
(D) Chloride

2015

45. Which physical characteristic of Romanowsky-type stains (e.g., Wright-Giemsa) makes them highly valuable for routine veterinary cytology?
(A) They excellently highlight cytoplasmic details and microorganisms.
(B) They provide excellent definition of nuclear chromatin but fail to stain cytoplasm.
(C) They selectively stain fat droplets within cells.
(D) They exclusively stain living cells and exclude dead ones.

NODIA

2017

46. During slide preparation for Fine Needle Aspiration Cytology (FNAC) of a solid mass, which technique is most appropriate for spreading a semi-solid or highly cellular aspirate?
(A) Starfish (needle-drag) preparation
(B) Blood smear (push slide) technique
(C) Squash (compression) preparation
(D) Line smear technique

2014

47. Based on cellularity and protein content, how is a pure transudate typically classified?
(A) Total protein > 3.0 g/dL and Total Nucleated Cell Count $> 5{,}000/\mu L$
(B) Total protein > 2.5 g/dL and Total Nucleated Cell Count $< 1{,}000/\mu L$
(C) Total protein < 2.5 g/dL and Total Nucleated Cell Count $> 5{,}000/\mu L$
(D) Total protein < 2.5 g/dL and Total Nucleated Cell Count $< 1{,}500/\mu L$

2015

48. In cytological evaluation, a lesion characterized by 80% degenerate neutrophils and 20% macrophages is best classified as which type of inflammation?
(A) Granulomatous
(B) Pyogranulomatous
(C) Suppurative (purulent)
(D) Eosinophilic

IV. Rubricyte

Which of the following represents the correct chronological sequence from most immature to most mature?

(A) II, IV, I, III
(B) I, II, III, IV
(C) II, I, IV, III
(D) IV, II, I, III

2011

71. Elevated levels of Glutamate Dehydrogenase (GLDH) in the serum of ruminants are highly specific for damage to which cellular structure?

(A) Biliary epithelium
(B) Hepatocyte mitochondria
(C) Pancreatic acinar cells
(D) Renal tubular epithelial cells

2007

72. Which process is responsible for the physiological elevation of Alkaline Phosphatase (ALP) commonly seen in young, growing puppies?

(A) Increased biliary excretion of hepatic enzymes
(B) Hepatic microsomal enzyme induction by steroids
(C) Osteoblastic activity in growing bones
(D) Intestinal absorption of colostral antibodies

2022

73. Which fraction of bilirubin is water-soluble and actively excreted into the biliary system by hepatocytes?

(A) Unconjugated bilirubin
(B) Biliverdin
(C) Urobilinogen
(D) Conjugated bilirubin

2021

74. In evaluating the diagnostic utility of Blood Urea Nitrogen (BUN), which of the following non-renal factors can significantly decrease BUN levels?

(A) Severe hepatic insufficiency
(B) High dietary protein intake
(C) Acute gastrointestinal hemorrhage
(D) Intense anaerobic physical exercise

2018

75. Exudative effusions in body cavities are primarily driven by which underlying pathophysiological mechanism?

(A) Decreased plasma oncotic pressure
(B) Increased vascular permeability
(C) Increased hydrostatic pressure
(D) Lymphatic obstruction

2024

76. In urinalysis, a positive dipstick reaction for occult blood with an absence of intact red blood cells on sediment evaluation implies the presence of:

(A) Active glomerular bleeding
(B) Hemoglobinuria or myoglobinuria
(C) Lower urinary tract infection
(D) Artifactual dipstick error due to high specific gravity

2018

77. Which cast found in urine sediment is indicative of chronic, severe renal tubular degeneration and represents the final stage of cast degradation?

(A) Waxy cast
(B) Hyaline cast
(C) Granular cast
(D) Cellular cast

2015

78. When performing a Baermann technique, what is the primary target organism sought in the feces?

(A) Tapeworm proglottids
(B) Protozoan cysts
(C) Nematode larvae
(D) Trematode eggs

2005

79. The Nonne-Apelt test performed on Cerebrospinal Fluid (CSF) is utilized specifically to detect an elevation in:

(A) Albumin
(B) Glucose
(C) Cholesterol
(D) Globulin

2015

80. Assess the validity of the following assertion and reason regarding canine urinalysis and choose the correct option:

Assertion (A): The presence of calcium oxalate monohydrate crystals in urine sediment is strongly suggestive of ethylene glycol toxicity.

Reason (R): Ethylene glycol is metabolized in the liver to oxalic acid, which readily binds calcium and precipitates in the renal tubules.

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

Click Here to Buy book on Amazon

2017

60. Select the correct sequential order of steps involved in routine tissue processing for histopathology after biopsy collection:
(A) Dehydration, Clearing, Fixation, Embedding, Sectioning
(B) Fixation, Dehydration, Clearing, Embedding, Sectioning
(C) Fixation, Clearing, Dehydration, Sectioning, Embedding
(D) Embedding, Fixation, Dehydration, Clearing, Sectioning

2019

61. Why is a 10% neutral buffered formalin ratio of 10:1 (formalin volume to tissue volume) universally recommended for biopsy fixation?
(A) To ensure complete, uniform tissue penetration
(B) To rapidly dissolve cellular lipids and clear tissue
(C) To decalcify microscopic bony spicules in soft tissue
(D) To selectively preserve glycogen stores in hepatocytes

2002

62. Which blood collection site is most appropriate for drawing large volumes of blood from an adult bovine for comprehensive clinical pathology testing?
(A) Cephalic vein
(B) Ear vein
(C) Jugular vein
(D) Saphenous vein

2012

63. What effect does delayed separation of serum from the cellular clot have on serum glucose concentration?
(A) Falsely elevated due to glycogenolysis
(B) Falsely elevated due to cellular lysis
(C) Unaffected as long as the tube remains sealed
(D) Falsely decreased due to cellular glycolysis

2022

64. When evaluating the erythrogram, what does an elevated Total Erythrocyte Count (TEC) with an elevated PCV and hemoglobin most likely indicate in a severely dehydrated animal?
(A) Primary absolute polycythemia (Polycythemia vera)
(B) Secondary absolute polycythemia
(C) Relative polycythemia
(D) Compensatory macrocytosis

2004

65. Howell-Jolly bodies, small spherical nuclear remnants found in erythrocytes, are most commonly observed in animals with which of the following conditions?
(A) Severe, chronic iron deficiency anemia
(B) Splenic dysfunction or splenectomy
(C) Acute, systemic lead toxicosis
(D) End-stage chronic renal disease

2010

66. A "stress leukogram" induced by endogenous corticosteroids is classically characterized by which specific profile?
(A) Neutropenia, lymphocytosis, and eosinophilia
(B) Neutrophilia, lymphocytosis, and basophilia
(C) Eosinophilia, monocytosis, and neutropenia
(D) Neutrophilia, lymphopenia, and eosinopenia

2004

67. Which automated hematology analyzer technology relies on the principle that cells pass through an aperture, briefly interrupting an electrical current to count and size them?
(A) Impedance technology
(B) Flow cytometry
(C) Laser light scattering
(D) Quantitative buffy coat analysis

2024

68. The appearance of "toxic change" in neutrophils is an indicator of accelerated myelopoiesis and systemic inflammation. Which of the following is a classic feature of toxic change?
(A) Nuclear hypersegmentation and chromatin condensation
(B) Complete loss of cytoplasmic primary granules
(C) Cytoplasmic vacuolation and Döhle bodies
(D) Frequent formation of nuclear Barr bodies

NODIA

2007

69. Babesia gibsoni is an important tick-borne hemoparasite of dogs. Where exactly does this parasite localize and replicate within the host?
(A) Inside erythrocytes
(B) Inside neutrophils
(C) Inside macrophages
(D) Freely in the blood plasma

2016

70. Consider the following erythrocyte maturation stages:
I. Metarubricyte
II. Rubriblast
III. Reticulocyte

2016

49. Which of the following is a recognized cytological criterion of malignancy in evaluating epithelial neoplasms?
(A) Uniform nuclear size
(B) Macronucleoli and multinucleation
(C) Low nucleus-to-cytoplasm (N:C) ratio
(D) Minimal mitotic figures

2016

50. Consider the following statements concerning urine specific gravity (USG):
I. USG is an assessment of the concentrating ability of the renal tubules.
II. Isosthenuria is defined by a USG range of 1.008 to 1.012.
III. Refractometry is less accurate than a dipstick for determining veterinary USG.
IV. Hyposthenuria always indicates terminal renal failure.
V. A USG of 1.045 in a dehydrated dog indicates appropriate renal compensation.
Which of the above statements are correct?
(A) I, II and IV
(B) I, II and V
(C) II, III, IV and V
(D) I, III and V

2025

51. The presence of ketones in a canine urine sample (ketonuria) on a dipstick test is most closely associated with which metabolic disruption?
(A) Altered carbohydrate and lipid metabolism
(B) Pathological skeletal protein catabolism
(C) Acute, massive hepatocellular necrosis
(D) Advanced primary hyperparathyroidism

2010

52. On microscopic examination of urine sediment, which crystal typically appears as a six-sided, colorless hexagon and is indicative of a specific metabolic defect?
(A) Calcium oxalate monohydrate
(B) Magnesium ammonium phosphate (Struvite)
(C) Cystine
(D) Ammonium biurate

2012

53. The presence of numerous cellular casts in urine sediment (cylindruria) is a specific indicator of disease primarily affecting which structure?
(A) Renal tubules
(B) Glomerulus
(C) Ureters
(D) Urinary bladder

2014

54. When testing feces for occult blood, the reaction is dependent on the pseudoperoxidase activity of which specific molecule?
(A) Myoglobin
(B) Cytochrome C
(C) Transferrin
(D) Hemoglobin

2025

55. Which flotation solution is conventionally favored for the detection of Giardia cysts in feces due to its specific gravity and minimal distortion of the cysts?
(A) Saturated sodium chloride
(B) Zinc sulfate solution
(C) Saturated sucrose solution
(D) Formalin-ether solution

2017

56. Deep skin scrapings that draw capillary blood are essential for the accurate diagnostic identification of which ectoparasite?
(A) Cheyletiella yasguri
(B) Otodectes cynotis
(C) Demodex canis
(D) Psoroptes ovis

2007

57. Which standard staining method is best utilized for microscopic examination of hair shafts suspected of dermatophyte infection (e.g., Microsporum canis)?
(A) Gram stain
(B) Romanowsky stain
(C) India ink
(D) Lactophenol cotton blue

2018

58. Pandy's test on Cerebrospinal Fluid (CSF) is utilized primarily to detect an abnormal increase in which specific component?
(A) Globulins
(B) Glucose
(C) Erythrocytes
(D) Lactate

2015

59. In the analysis of CSF, a marked increase in the nucleated cell count characterized predominantly by neutrophils is clinically termed:
(A) Mononuclear pleocytosis
(B) Suppurative pleocytosis
(C) Eosinophilic pleocytosis
(D) Xanthochromia

Downloaded from www.pdf.tube

2009

81. A core biopsy of the liver is typically preferred over a fine needle aspirate (FNA) when attempting to accurately diagnose which condition?
(A) Hepatic lipidosis
(B) Hepatic fibrosis
(C) Lymphoma infiltration
(D) Suppurative hepatitis

2017

82. Which cellular component is evaluated using the New Methylene Blue (NMB) stain to definitively identify reticulocytes in a blood sample?
(A) Mitochondrial remnants
(B) Nuclear fragments
(C) Denatured hemoglobin
(D) Precipitated ribosomal RNA

2007

83. The clinical condition 'azotemia' is exclusively defined by laboratory findings of:
(A) Elevated BUN and creatinine
(B) Decreased urine specific gravity
(C) Increased blood ammonia levels
(D) Presence of protein and glucose in urine

2019

84. Which serum protein is specifically recognized as a major negative acute phase protein, meaning its concentration decreases during acute inflammation?
(A) Haptoglobin
(B) C-reactive protein
(C) Albumin
(D) Serum amyloid A

2015

85. The term 'dysproteinemia' accurately describes which of the following clinical scenarios?
(A) Altered concentrations of specific serum proteins
(B) Normal protein levels with equal albumin and globulin
(C) Absolute loss of protein through the renal tubules
(D) Complete failure of the liver to synthesize urea

2001

86. Which electrolyte imbalance is classically associated with ruptured urinary bladder or urethral obstruction in cats?
(A) Hypercalcemia
(B) Hypokalemia
(C) Hyponatremia
(D) Hyperkalemia

2007

87. A modified transudate typically forms under which primary physiological derangement?
(A) Massive systemic inflammation increasing capillary permeability
(B) Chronic increases in hydrostatic pressure with protein addition
(C) Acute blood vessel rupture directly into the abdominal cavity
(D) Severe hypoproteinemia resulting in generalized fluid shifting

2009

88. When cytologically differentiating a benign from a malignant neoplasm, which feature is generally considered indicative of a benign process?
(A) Frequent and abnormal mitotic figures
(B) High and variable nucleus-to-cytoplasm (N:C) ratio
(C) Uniform population of cells with a low N:C ratio
(D) Marked anisokaryosis and multinucleation

2017

89. Urine pH measurement via a dipstick can be significantly altered by post-collection factors. Which handling error falsely increases urine pH?
(A) Storing the sample tightly sealed at 4°C
(B) Contamination with exogenous ascorbic acid
(C) Rapid centrifugation before dipping the stick
(D) Leaving the sample unsealed at room temperature

NODIA

2010

90. Match the crystal type found in urine in List I with its characteristic morphology in List II:

List I: (Crystal Type)
P. Ammonium biurate
Q. Calcium oxalate dihydrate
R. Bilirubin
S. Cystine

List II: (Morphology)
1. Hexagonal plates
2. Thorn apple shape (brown spheres with spicules)
3. Small square envelopes with an 'X' inside
4. Yellow-brown needle-like bundles

(A) P-2, Q-3, R-4, S-1
(B) P-3, Q-1, R-2, S-4
(C) P-1, Q-4, R-3, S-2
(D) P-2, Q-4, R-1, S-3

2007

91. The macroscopic physical property of urine described as "flocculent" most specifically implies the presence of:

dark purple granules

2002

133. A "right shift" in a leukogram refers to an increase in the number of which specific circulating cells?
(A) Hypersegmented neutrophils
(B) Immature band neutrophils
(C) Activated reactive lymphocytes
(D) Immature metamyelocytes

2017

134. When interpreting a platelet count, the term "Mean Platelet Volume" (MPV) provides clinically useful information regarding:
(A) The lifespan and destruction rate of circulating platelets
(B) The total relative mass of megakaryocytes in the bone marrow
(C) The intrinsic aggregation capability and function of platelets
(D) Accelerated thrombopoiesis, as new platelets are larger

2011

135. The hemoparasite Trypanosoma evansi is responsible for causing which major disease in horses and camels?
(A) East Coast Fever
(B) Surra
(C) Tick Fever
(D) Heartwater

2015

136. Where is the hemoparasite Trypanosoma evansi located when observing a peripheral blood smear?
(A) Intracellularly inside the erythrocyte
(B) Intracellularly inside the mononuclear cells
(C) Extracellularly in the plasma
(D) Intracellularly inside the platelets

2010

137. Which combination of biochemical findings is most strongly indicative of severe muscle trauma or rhabdomyolysis?
(A) High ALT and High ALP
(B) Low BUN and High SDMA
(C) High GGT and High Bilirubin
(D) High AST and High CK

2015

138. In a healthy animal, the vast majority of bilirubin circulating in the blood prior to hepatic uptake is in which form?
(A) Unconjugated bilirubin bound to albumin
(B) Conjugated bilirubin bound to serum globulin
(C) Free, unconjugated biliverdin in plasma
(D) Free circulating urobilinogen molecules

2004

139. Which of the following is considered an artifactual cause of apparent hypoproteinemia when measured via a refractometer?
(A) Severe sample hemolysis causing red cell lysis
(B) An incorrectly calibrated refractometer prism
(C) Moderate to severe lipemia in a postprandial sample
(D) Severe hyperglycemia elevating plasma osmolarity

2010

140. Which sequence correctly represents the proper order of steps for processing a sample for fine needle aspiration (FNA) cytology?
(A) Isolate mass → Insert needle with negative pressure → Release pressure → Withdraw needle → Expel onto slide
(B) Isolate mass → Insert needle → Expel onto slide → Apply negative pressure → Withdraw needle
(C) Isolate mass → Apply negative pressure → Insert needle → Withdraw needle → Release pressure
(D) Insert needle → Isolate mass → Withdraw needle → Apply negative pressure → Expel onto slide

2007

141. Epithelial cells on a cytological preparation typically exhibit which characteristic arrangement?
(A) Cohesive clusters or sheets with distinct cell borders
(B) Individual, scattered spindle-shaped cells with cytoplasmic tails
(C) Highly individual, round cells lacking any cell-to-cell adhesion
(D) Long, parallel streams of fusiform cells with poorly defined borders

2009

142. A cytological sample showing a highly cellular population of discrete, individually arranged cells with distinct borders and round nuclei is most characteristic of which tumor type?
(A) Epithelial carcinoma
(B) Mesenchymal sarcoma
(C) Round cell tumor

Click Here to Buy book on Flipkart

(B) Freely moving in the cytoplasm
(C) Attached to the external membrane
(D) At the periphery of the cell

2009

103. Which specific liver enzyme is bound to the canalicular membrane of hepatocytes and is considered a primary indicator of cholestasis?
(A) ALT
(B) ALP
(C) AST
(D) CK

2005

104. Which of the following best describes the physiological basis for measuring pre- and post-prandial serum bile acids?
(A) Fasting allows bile acids to accumulate inside the vascular space, while feeding triggers rapid excretion into the urinary system.
(B) Hepatocytes synthesize bile acids exclusively in response to food intake, keeping the pre-prandial concentrations completely undetectable.
(C) Gallbladder contraction releases bile acids, which are then cleared from portal blood by a healthy liver.
(D) Ingestion of nutrients directly blocks the hepatic pathways responsible for the baseline synthesis of all major bile acid compounds.

2002

105. Hypercholesterolemia in the dog is most frequently identified as a secondary complication to which endocrine disorder?
(A) Hyperthyroidism
(B) Hypoadrenocorticism
(C) Hyperparathyroidism
(D) Hypothyroidism

2009

106. In assessing kidney function, an increase in SDMA occurs at what approximate percentage of functional nephron loss?
(A) 25%
(B) 50%
(C) 75%
(D) 90%

2022

107. Which of the following is considered a primary cause of post-renal azotemia?
(A) Severe dehydration
(B) Urethral obstruction
(C) Primary glomerular disease
(D) Acute tubular necrosis

2015

108. The measurement of serum fructosamine is primarily used in veterinary clinical pathology to monitor which condition?
(A) Exocrine pancreatic enzyme insufficiency
(B) Glycemic control in diabetes mellitus
(C) Chronic progressive kidney disease
(D) Hepatic lipidosis and liver failure

2009

109. What is the expected alteration in serum total protein and PCV in an animal experiencing severe acute dehydration?
(A) Increased Total Protein, Increased PCV
(B) Decreased Total Protein, Decreased PCV
(C) Normal Total Protein, Increased PCV
(D) Increased Total Protein, Decreased PCV

2010

110. Consider the following statements regarding serum electrolyte evaluation:
I. Sodium is the primary determinant of extracellular fluid osmolality.
II. Hemolysis of a blood sample typically causes artifactual hypokalemia in all species.
III. Chloride levels generally rise and fall proportionally with sodium levels.
IV. PTH and Calcitriol are major regulators of serum calcium and phosphorus.
Which of the above statements is/are INCORRECT?
(A) I and IV
(B) II only
(C) II and III
(D) I, III and IV

2002

111. Which acid-base disorder is characterized by a primary decrease in blood pH and a marked decrease in serum bicarbonate?
(A) Respiratory acidosis
(B) Metabolic alkalosis
(C) Respiratory alkalosis
(D) Metabolic acidosis

2013

112. The term "exfoliative cytology" is best applied to the examination of cells obtained via which method?
(A) Performing a core needle biopsy of deep solid organs
(B) Complete surgical excision and histopathology of a mass
(C) Collecting naturally exfoliated or scraped cells
(D) Harvesting cellular bone marrow spicules

Downloaded from www.pdf.tube

(A) High levels of dissolved glucose
(B) Suspended particulate matter
(C) Normal equine mucus production
(D) Elevated levels of bilirubin

2006

92. Identify the optimal fixative utilized specifically when preparing a tissue sample for standard electron microscopic examination, rather than light microscopy.
(A) 10% Neutral Buffered Formalin
(B) Glutaraldehyde
(C) Bouin's solution
(D) Absolute Ethanol

2023

93. A wedge biopsy is generally advantageous over a punch biopsy because it:
(A) Provides a larger sample showing the margin between normal and abnormal tissue
(B) Avoids the clinical requirement for either local infiltration or general anesthesia
(C) Safely collects only the most superficial epidermal layers to minimize hemorrhage
(D) Is performed exclusively through a highly automated, spring-loaded needle system

2006

94. The specific term used to describe a reduction in the total number of circulating leukocytes below the physiological reference range is:
(A) Leukocytosis
(B) Neutrophilia
(C) Leukopenia
(D) Pancytopenia

2009

95. What is the fundamental difference between plasma and serum?
(A) Serum contains active coagulation factors, while plasma does not.
(B) Plasma is harvested from clotted blood, while serum requires heparin.
(C) Serum has a much higher concentration of erythrocytes than plasma.
(D) Plasma contains fibrinogen, while serum does not have fibrinogen.

2001

96. Which artifact is reliably prevented by fasting an animal for 12 hours prior to a routine biochemical blood draw?
(A) Icterus
(B) Hemolysis
(C) Lipemia
(D) Polychromasia

2001

97. The presence of macrocytic, hypochromic erythrocytes on a hemogram most strongly points toward which underlying clinical process?
(A) Severe, long-standing iron deficiency
(B) Marked erythroid regeneration
(C) Acute, non-regenerative anemia
(D) Primary bone marrow failure

2014

98. Polychromasia observed on a Romanowsky-stained blood smear corresponds to which specific cell type identified using New Methylene Blue stain?
(A) Spherocyte
(B) Heinz body erythrocyte
(C) Megakaryocyte
(D) Reticulocyte

NODIA

2015

99. A peripheral blood smear containing numerous target cells (codocytes) is frequently associated with which organ dysfunction in dogs?
(A) Liver disease
(B) Cardiac failure
(C) Renal failure
(D) Pancreatitis

2024

100. Consider the following statements regarding leukocyte evaluation:
I. Eosinophilia is often associated with allergic reactions and parasitism.
II. Monocytosis is an indicator of acute, overwhelming viral infection exclusively.
III. Lymphopenia is a classic component of the stress leukogram.
Which of the above statements are correct?
(A) I and II
(B) I and III
(C) II and III
(D) I only

2006

101. Which condition is characterized by a pathological decrease in circulating platelet numbers?
(A) Thrombocytopenia
(B) Thrombocytosis
(C) Pancytopenia
(D) Leukopenia

2025

102. Anaplasma marginale, a vital hemoparasite of cattle, is characteristically located in which position within the infected erythrocyte?
(A) In the central region of the cell

(C) Demodex canis
(D) Psoroptes cuniculi

2016

123. Normal cerebrospinal fluid (CSF) in domestic animals is physically characterized as:
(A) Yellow and turbid
(B) Colorless and clear
(C) Pinkish and slightly viscous
(D) Opaque white

2023

124. The term "xanthochromia" when describing CSF refers to which physical appearance?
(A) A milky white or opalescent appearance due to lipid
(B) A yellowish discoloration from prior hemorrhage
(C) A bright red or pinkish color from acute iatrogenic bleeding
(D) A cloudy or turbid appearance due to bacterial overgrowth

2017

125. Which diagnostic test is the absolute minimum requirement to definitively differentiate between an acute inflammatory skin lesion and a neoplastic skin mass?
(A) Fine needle aspiration or biopsy
(B) Comprehensive complete blood count
(C) Multi-parameter serum biochemistry
(D) Routine multi-stick urinalysis

2022

126. A 10 mL sample of normal canine blood with a PCV of 45% should yield approximately what volume of serum after complete clotting and centrifugation?
(A) 1.0 to 1.5 mL
(B) 2.0 to 3.0 mL
(C) 4.5 to 5.5 mL
(D) 8.0 to 9.0 mL

2015

127. Why should EDTA NOT be used as an anticoagulant when submitting a sample for a comprehensive serum biochemistry profile?
(A) It irreversibly degrades glucose molecules in the plasma
(B) It causes rapid precipitation of total serum proteins
(C) It induces very rapid degradation of key liver enzymes
(D) It causes severe hypocalcemia and hyperkalemia

2004

128. What is the specific clinical implication of an elevated Mean Corpuscular Volume (MCV) coupled with a normal Mean Corpuscular Hemoglobin Concentration (MCHC)?
(A) Microcytic, normochromic anemia
(B) Normocytic, hypochromic anemia
(C) Macrocytic, normochromic anemia
(D) Macrocytic, hypochromic anemia

2022

129. Rouleaux formation, characterized by the stacking of erythrocytes like coins, is considered a normal finding in the blood smears of which species?
(A) Dog
(B) Horse
(C) Cow
(D) Pig

2003

130. Consider the following steps for preparing and examining a standard blood smear:
I. Place a small drop of blood near the frosted edge of the slide.
II. Allow the smear to air dry rapidly.
III. Use a spreader slide at a 30-45 degree angle to push the blood forward.
IV. Stain the slide using a Romanowsky stain.
What is the correct sequence of these steps?
(A) I, III, II, IV
(B) III, I, II, IV
(C) I, II, III, IV
(D) I, IV, III, II

2024

131. Which white blood cell is primarily responsible for parasitic defense and modulating allergic reactions, identifiable by its bright red/orange cytoplasmic granules?
(A) Eosinophil
(B) Neutrophil
(C) Basophil
(D) Monocyte

2007

132. During manual differential leukocyte counting, what distinct morphological feature identifies a monocyte?
(A) Small size, extremely sparse blue cytoplasm, and a dense round nucleus
(B) Multi-lobulated nucleus with extremely clear to very pale pink cytoplasm
(C) Large size, vacuolated blue-gray cytoplasm, and lobulated nucleus
(D) Bi-lobed nucleus packed with very dense and

Click Here to Buy book on Amazon

from a patient

2024

113. During cytological assessment of a lymph node aspirate, a predominance of small, mature lymphocytes ($> 80\%$) with scattered prolymphocytes and macrophages most likely indicates:
(A) Lymphoma
(B) Normal or reactive lymph node
(C) Metastatic carcinoma
(D) Pyogranulomatous lymphadenitis

2008

114. Which specific finding in a body cavity effusion is highly characteristic of feline infectious peritonitis (FIP)?
(A) A pure transudate characterized by extremely low protein and low cellularity
(B) A highly cellular sample with predominantly malignant, pleomorphic epithelial cells
(C) A milky, chylous effusion packed with a high population of small lymphocytes
(D) A viscous, high-protein exudate with non-degenerate neutrophils

2023

115. Cytologically, what characterizes a mast cell tumor aspirate?
(A) Round cells containing distinct purple intracytoplasmic granules
(B) Spindle-shaped mesenchymal cells producing pink extracellular matrix
(C) Cohesive clusters of polygonal epithelial cells with prominent desmosomes
(D) Large activated macrophages containing negatively-staining fungal bodies

2006

116. A high-protein diet in a normal, healthy dog or cat will typically cause the urine pH to be:
(A) Highly alkaline (pH > 8.0)
(B) Exactly neutral (pH 7.0)
(C) Acidic (pH 5.5 to 7.0)
(D) Highly variable depending on the time of day

2004

117. Urine chemical reagent strip pads for detecting sugar utilize an enzymatic reaction that is highly specific for:
(A) Glucose
(B) Fructose
(C) Galactose
(D) Lactose

2008

118. Which specific cast type observed in urine sediment is composed of a mucoprotein matrix (Tamm-Horsfall protein) without any cellular inclusions, and can be seen in low numbers in normal animals?
(A) Epithelial cast
(B) Erythrocyte cast
(C) Waxy cast
(D) Hyaline cast

2013

119. A microscopic fecal examination reveals large, heavy ova with distinct bipolar plugs. This morphology is diagnostic for which parasite?
(A) Toxocara canis
(B) Trichuris vulpis
(C) Ancylostoma caninum
(D) Dipylidium caninum

2005

120. Consider the following statements regarding biopsy collection and tissue processing:
I. A punch biopsy is primarily utilized for taking small cylindrical cores of skin.
II. Excised tissues should be placed in formalin at a ratio of 1:1.
III. Tissues thicker than 1 cm should be incised or "loafed" prior to fixation to ensure adequate penetration.
IV. Autolysis begins immediately after tissue removal and is halted by proper fixation.
Which of the above statements is/are INCORRECT?
(A) I and II
(B) II only
(C) III and IV
(D) I, III and IV

NODIA

2007

121. To properly evaluate maldigestion related to exocrine pancreatic insufficiency via fecal examination, which stain is applied to detect undigested fat (steatorrhea)?
(A) Lugol's iodine
(B) New Methylene Blue
(C) Eosin
(D) Sudan III or IV

2010

122. Which ectoparasite is classically located on the surface of the skin and hair coat, walking rapidly, and is commonly referred to as "walking dandruff"?
(A) Cheyletiella spp.
(B) Sarcoptes scabiei

(D) Benign adenoma

2013

143. Which type of epithelial cell is typically the largest found in urine sediment, possessing a small central nucleus and angular, folded edges?
(A) Renal tubular epithelial cell
(B) Transitional epithelial cell
(C) Macrophage
(D) Squamous epithelial cell

2017

144. In urinalysis, the presence of specific gravity of 1.010 with concurrent severe dehydration is termed:
(A) Hyposthenuria
(B) Hypersthenuria
(C) Isosthenuria
(D) Normosthenuria

2007

145. What is the clinical significance of finding numerous transitional epithelial cells displaying marked anaplasia and mitotic figures in a canine urine sample?
(A) Normal sloughing from the bladder wall
(B) Transitional cell carcinoma (TCC)
(C) Catheterization artifact
(D) Acute bacterial cystitis

2007

146. A patient presenting with polyuria and polydipsia has a persistently low urine specific gravity (1.004) and a complete lack of response to water deprivation testing. This is characteristic of:
(A) Diabetes mellitus
(B) Chronic renal failure
(C) Primary hyperparathyroidism
(D) Diabetes insipidus

2014

147. Which fecal examination method is specifically designed to concentrate trematode (fluke) eggs, taking advantage of their high specific gravity?
(A) Fecal sedimentation
(B) Simple flotation
(C) Centrifugal flotation with sucrose
(D) Baermann technique

2002

148. Sarcoptes scabiei in dogs typically presents clinically with intense pruritus. Which morphological feature aids in its microscopic identification?
(A) Long, jointed pedicels with bell-shaped suckers
(B) Cigar-shaped body with stumpy legs
(C) Long, unjointed pedicels with terminal suckers
(D) Large size visible to the naked eye

2011

149. Why is a wedge biopsy specifically advantageous for histopathological evaluation of an ulcerative dermal lesion?
(A) It allows evaluation of the transition zone from normal tissue to the ulcer edge.
(B) It completely eliminates the need for chemical or formalin fixation in most clinical cases.
(C) It successfully removes the entire tumor with wide three-centimeter margins in every case.
(D) It requires only a very superficial scrape of the epidermal layers without deep incision.

2006

150. Given below are two statements, one labeled as Assertion (A) and the other as **Reason (R):**
Assertion (A): The measurement of Total Leukocyte Count (TLC) alone is insufficient to accurately assess an animal's inflammatory status.
Reason (R): A normal TLC can hide severe, potentially life-threatening shifts in specific leukocyte populations (e.g., neutropenia with a severe left shift masked by a concurrent lymphocytosis).
Which of the following is correct?
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2018

151. Which factor is most critical when preserving tissue samples for accurate immunohistochemistry (IHC) later?
(A) Freezing the tissue sample at -20°C immediately following surgical removal
(B) Fixation in 10% neutral buffered formalin without over-fixation
(C) Placing the fresh tissue sample directly into concentrated hydrochloric acid
(D) Leaving the tissue to air-dry completely on a glass slide for twenty-four hours

Downloaded from www.pdf.tube

high specific gravities and high osmotic pressures that instantly destroy the delicate trophozoite morphology.

Which of the following is correct?

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2006

209. The presence of profound hypocalcemia commonly observed in lactating dairy cattle (Parturient Paresis) directly results in which prominent clinical sign?

(A) Tetanic muscle spasms and tetany
(B) Flaccid paralysis
(C) Severe bleeding and hemorrhagic diathesis
(D) Profound polyuria and urinary incontinence

2005

210. Match the cellular inclusions or structural abnormalities in List I with their corresponding primary compositions or causes in List II:

List I: (Cellular Inclusion or Abnormality)

P. Heinz body
Q. Howell-Jolly body
R. Basophilic stippling
S. Döhle body

List II: (Composition or Cause)

1. Aggregated ribosomal RNA within a toxic neutrophil
2. Denatured and precipitated hemoglobin in an erythrocyte
3. Nuclear (DNA) remnant in an erythrocyte
4. Aggregated ribosomes/RNA in an erythrocyte indicating regeneration or lead toxicity

(A) P-2, Q-3, R-4, S-1
(B) P-3, Q-2, R-1, S-4
(C) P-1, Q-4, R-2, S-3
(D) P-4, Q-1, R-3, S-2

NODIA

****

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

2008

161. A 7-year-old cat presents with a prolonged activated partial thromboplastin time (aPTT) and a normal prothrombin time (PT). Which coagulation factor is most likely deficient?
(A) Factor VII
(B) Factor VIII
(C) Factor X
(D) Factor II

2011

162. Which of the following is considered a primary indicator of skeletal muscle damage in domestic animals?
(A) Alkaline Phosphatase
(B) Gamma-Glutamyl Transferase
(C) Lipase
(D) Creatine Kinase (CK)

2004

163. The primary source of serum amylase in the dog is the pancreas, but which organ is responsible for its clearance, thereby elevating serum levels when diseased?
(A) Kidney
(B) Liver
(C) Small Intestine
(D) Spleen

2024

164. What is the clinical implication of a severe decrease in serum albumin levels (hypoalbuminemia) to below 1.5 g/dL?
(A) Rapid development of severe systemic hypertension
(B) Acute cardiac arrest due to lack of calcium binding sites
(C) Loss of oncotic pressure causing edema or effusions
(D) Spontaneous arterial thrombosis due to low antithrombin III

2016

165. During a routine serum biochemistry, artifactual hyperkalemia and profound hypocalcemia can occur if blood is drawn into which incorrect tube?
(A) K3-EDTA tube
(B) Lithium heparin tube
(C) Plain red top tube
(D) Serum separator tube (SST)

2013

166. Which cytological preparation technique is specifically recommended for evaluating fluids with very low cellularity, such as normal CSF?
(A) Direct smear
(B) Squash preparation
(C) Starfish preparation
(D) Cytocentrifugation

2011

167. A pyogranulomatous inflammatory response on cytology is defined by the significant co-presence of which two cell types?
(A) Neutrophils and Lymphocytes
(B) Neutrophils and Macrophages
(C) Eosinophils and Basophils
(D) Lymphocytes and Plasma cells

2015

168. Which specific cytological feature strongly suggests a malignant mesenchymal tumor (sarcoma) over a benign fibroma?
(A) Uniformly arranged spindle-shaped cells with low pleomorphism
(B) Very distinct cell borders paired with abundant mature collagen
(C) Marked anisokaryosis, multiple nucleoli, and mitotic figures
(D) A complete and consistent absence of all inflammatory cells

2015

169. What is the underlying cause for the formation of a hyaline cast within the renal tubules?
(A) Accumulation of desquamated epithelial cells in glomeruli
(B) Extensive colonization by bacteria forming a dense matrix
(C) Sudden crystallization of uric acid within renal tubules
(D) Precipitation of Tamm-Horsfall mucoprotein in acid urine

2008

170. Consider the following statements concerning urinary crystals:
I. Struvite crystals often form in alkaline urine and are associated with urease-producing bacterial infections in dogs.
II. Uric acid crystals are a normal finding in the urine of healthy Dalmatians.
III. Calcium oxalate dihydrate crystals definitively diagnose ethylene glycol toxicity.
IV. Bilirubin crystals are never a normal finding in healthy canine urine.
Which of the above statements is/are correct?
(A) I and II
(B) II and III
(C) I, II and IV

Click Here to Buy book on Flipkart

(D) III and IV

2007

171. Microscopic examination of urine sediment reveals motile, pear-shaped organisms with undulating membranes and flagella. This is diagnostic for:
(A) Capillaria plica ova
(B) Trichomonas spp.
(C) Dioctophyma renale ova
(D) Giardia lamblia cysts

2005

172. The presence of numerous fat droplets in the fecal smear of a dog is clinically termed:
(A) Amylorrhea
(B) Steatorrhea
(C) Creatorrhea
(D) Melena

2002

173. Which specific physical characteristic of a feces sample directly implies upper gastrointestinal bleeding (e.g., gastric ulcer)?
(A) Black, tarry appearance (Melena)
(B) Frank red blood on the surface (Hematochezia)
(C) Pale, clay-colored appearance (Acholic)
(D) Extremely watery with mucus

2001

174. To definitively diagnose a dermatophyte infection via culture, which specific growth medium is classically employed?
(A) Blood agar
(B) MacConkey agar
(C) Dermatophyte Test Medium (DTM)
(D) Sabouraud Dextrose Agar without cycloheximide

2023

175. During biopsy collection, what is the major detrimental consequence of freezing a tissue sample before placing it in formalin?
(A) Immediate loss and denaturation of all structural protein elements
(B) Complete conversion of tissue into an unrecognizable liquid state
(C) Selective destruction of cell nuclei while fully preserving cytoplasm
(D) Artifactual ice crystal formation disrupting cells

NODIA

2023

176. What is the fundamental principle driving specific gravity measurement via a veterinary refractometer?
(A) The physical mass of the liquid directly pushing a highly calibrated mechanical weight
(B) The electrical conductivity and ion flow measured within the urine sample matrix
(C) Refraction of light through a liquid proportional to dissolved solute concentration
(D) The chemical reaction and staining of dissolved ions with the surface prism glass

2021

177. When diagnosing an immune-mediated hemolytic anemia (IMHA) in a dog, which combination of hematological findings is most pathognomonic?
(A) Marked microcytosis, hypochromasia, and reactive thrombocytosis
(B) Spherocytosis, autoagglutination, and a positive Coombs test
(C) Multiple Heinz bodies, eccentrocytes, and acute methemoglobinemia
(D) Prominent schistocytes, thrombocytopenia, and a prolonged aPTT

2022

178. What specific clinical interpretation is made when a high percentage of target cells is noted on a canine blood smear?
(A) Immediate morphological precursor to total erythrocyte lysis
(B) Simple in vitro artifact of excessive EDTA anticoagulant
(C) Specific pathognomonic indicator of systemic lead poisoning
(D) Increased membrane lipid ratio from liver disease

2025

179. A marked eosinophilia is most consistently observed in animals suffering from which type of pathological insult?
(A) Parasitic migrations and hypersensitivity reactions
(B) Acute bacterial sepsis with massive endotoxin release
(C) Severe systemic viral infections of sudden onset
(D) Severe physical trauma causing extensive necrosis

2014

180. Consider the following statements regarding thrombocytopenia:

I. It is the most common acquired hemostatic disorder in domestic animals.
II. It typically manifests clinically as large, deep tissue hematomas.
III. It can be caused by decreased production, increased consumption, or immune-mediated destruction.
IV. Clinical signs usually do not appear until the platelet count drops below 30,000 to 50,000/μL.

Which of the above statements is/are INCORRECT?
(A) I and III
(B) II only
(C) III and IV
(D) I, II and IV

2016
181. In the evaluation of bone marrow aspirates, the Myeloid to Erythroid (M:E) ratio is used to determine:
(A) Granulocytic precursor to nucleated erythroid precursor ratio
(B) The absolute number of circulating mature leukocytes in peripheral blood
(C) The specific ratio of tissue macrophages to mature erythrocytes in marrow
(D) The exact rate of active iron incorporation into newly formed hemoglobin

2009
182. A 3-year-old dog presents with acute bleeding. Laboratory results show a prolonged PT, prolonged aPTT, severe thrombocytopenia, and elevated fibrin degradation products (FDPs). What is the definitive diagnosis?
(A) Congenital Hemophilia A deficiency
(B) Inherited Von Willebrand Disease
(C) Vitamin K Antagonist Toxicity
(D) Disseminated Intravascular Coagulation

2012
183. Which specific electrolyte abnormality is the classic hallmark of hypoadrenocorticism (Addison's disease) in dogs?
(A) Hypernatremia and Hypokalemia
(B) Hyponatremia and Hyperkalemia
(C) Hypercalcemia and Hypophosphatemia
(D) Hypochloremia and Hypercalcemia

2004
184. In a clinical setting, measurement of the Anion Gap is primarily used to differentiate between causes of:
(A) Respiratory alkalosis
(B) Respiratory acidosis
(C) Metabolic acidosis
(D) Metabolic alkalosis

2021
185. When collecting a fluid sample from a suspected chylous effusion in the thorax, which diagnostic finding is most confirmatory?
(A) Cholesterol concentration in the fluid significantly higher than in the serum
(B) Presence of highly malignant epithelial cells
(C) Extremely low protein content (< 1.0 g/dL)
(D) Triglyceride concentration in the fluid significantly higher than in the serum

2002
186. Which stain is uniquely preferred for demonstrating the presence of occult lipid (fat) droplets within hepatocytes on a liver aspirate?
(A) Sudan IV
(B) Wright-Giemsa
(C) Periodic Acid-Schiff (PAS)
(D) Gram stain

NODIA

2007
187. During normal physical examination of a dog's urine, a strong "fruity" or "sweet" odor is a classic indicator of:
(A) Pseudomonas infection
(B) Ketonuria
(C) Severe proteinuria
(D) High urea concentration

2003
188. Which method relies strictly on the principle of immunochromatography (lateral flow technology) to detect the presence of specific infectious antigens in a clinical sample?
(A) Real-time PCR assays
(B) Rapid lateral flow immunochromatographic strip tests
(C) Agar gel immunodiffusion
(D) Laser flow cytometry

2002
189. The presence of numerous squamous epithelial cells in a free-catch (voided) urine sample is most correctly interpreted as:
(A) Normal distal urethra or genital tract contamination
(B) Highly indicative of severe renal tubular damage
(C) Definitively diagnostic for a transitional cell carcinoma
(D) A highly specific sign of acute bacterial cystitis

elevated hemoglobin value, which subsequently distorts calculated erythrocyte indices.

10. (A) Both A and R are true and R is the correct explanation of A.
Erythrocytes contain intracellular enzymes in concentrations many times higher than those found in normal serum. When red blood cell membranes rupture, these enzymes, particularly lactate dehydrogenase, escape directly into the extracellular fluid. Consequently, even mild hemolysis during collection or processing falsely elevates the measured serum enzyme activity, making clinical interpretation inaccurate.

11. (B) Nuclear streaming and cellular distortion
Applying excessive physical pressure when spreading an aspirate ruptures fragile cell membranes, which releases naked chromatin. This chromatin stretches across the slide, producing long, basophilic lines known as nuclear streaming. Cellular distortion and lysis prevent the evaluation of intact cell morphology, making it difficult to distinguish neoplastic from inflammatory processes.

12. (B) $\frac{\text{Hemoglobin}}{PCV} \times 100$
The mean corpuscular hemoglobin concentration represents the average concentration of hemoglobin in a specific volume of packed red blood cells. It is mathematically calculated by dividing the measured hemoglobin concentration by the packed cell volume and multiplying the result by one hundred. This index is expressed as grams of hemoglobin per deciliter of erythrocytes.

13. (A) Regenerative anemia
Regenerative anemia is characterized by the release of immature erythrocytes, specifically reticulocytes, from the bone marrow. Reticulocytes are larger than mature red blood cells, which increases the mean corpuscular volume. Because these young cells are still actively synthesizing hemoglobin, they contain less hemoglobin relative to their size, decreasing the mean cell hemoglobin concentration.

14. (C) Variation in cell size
Anisocytosis refers to an abnormal variation in the size of erythrocytes present on a blood film. This variation is commonly quantified by the red cell distribution width in automated analyzers. High anisocytosis is typical during regenerative responses where large reticulocytes circulate alongside normal mature cells, or in nutritional deficiencies like iron deficiency.

15. (D) Immune-mediated destruction of erythrocytes
Spherocytes form when macrophages in the spleen partially phagocytize antibody-coated red blood cells, removing a portion of the cell membrane. The remaining membrane seals itself, but because it has lost surface area relative to its volume, the cell assumes a sphere shape. This morphology is highly characteristic of immune-mediated hemolytic anemia.

16. (C) Heinz bodies
Heinz bodies represent clumps of denatured, precipitated hemoglobin attached to the inner erythrocyte membrane, caused by oxidative damage to the globin chains. Oxidative agents, such as those found in onions, garlic, or acetaminophen, overwhelm the cellular protective pathways. These inclusions are visible as round, protruding structures using new methylene blue staining.

17. (B) Band and immature neutrophils
A left shift indicates that the bone marrow is releasing immature granulocytes into peripheral blood in response to inflammatory signals. Band neutrophils, which lack the segmented nuclear lobes of mature cells, are the most common immature forms observed. The severity of the left shift reflects the intensity and urgency of the systemic inflammatory stimulus.

18. (D) Immature neutrophils outnumber mature neutrophils, usually with leukopenia.
A degenerative left shift occurs when the demand for neutrophils at the site of inflammation exceeds the production and storage capacity of the bone marrow. As a result, the number of immature band cells in circulation surpasses the mature forms, often accompanied by a low total white blood cell count, indicating a poor prognosis.

19. (A) Epinephrine
Epinephrine release during excitement, fear, or vigorous exercise triggers a rapid shift of leukocytes from the marginal pool to the circulating pool. This physiological response increases blood flow, washing neutrophils and lymphocytes back into circulation within minutes. It is characterized by a transient neutrophilia and lymphocytosis without a left shift in healthy animals.

Downloaded from www.pdf.tube

blood, without a concurrent reticulocytosis (appropriate regeneration), suggest?
(A) A completely normal, healthy physiological state in a dog
(B) Bone marrow injury, splenic disease, or lead toxicity
(C) Severe iron deficiency anemia
(D) Acute systemic dehydration

2001

200. Given below are two statements, one labeled as Assertion (A) and the other as **Reason (R):**
Assertion (A): Elevated serum amylase and lipase are not definitively diagnostic for acute pancreatitis in dogs.
Reason (R): Amylase and lipase are also produced by extrapancreatic tissues and their renal clearance is reduced in kidney disease, leading to non-specific elevations.
Which of the following is correct?
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2015

201. Which liver enzyme is induced by endogenous or exogenous glucocorticoids in the dog, creating a specific "steroid-induced" isoenzyme?
(A) Alanine Aminotransferase (ALT)
(B) Aspartate Aminotransferase (AST)
(C) Gamma-Glutamyl Transferase (GGT)
(D) Alkaline Phosphatase (ALP)

2004

202. Measurement of which specific serum parameter is the most sensitive and direct indicator of the depletion of body iron stores?
(A) Serum Ferritin
(B) Serum Iron
(C) Total Iron Binding Capacity (TIBC)
(D) Hemoglobin concentration

2003

203. Which fluid abnormality is definitively characterized by a high nucleated cell count ($> 5{,}000/\mu L$), a high total protein (>3.0 g/dL), and the presence of degenerate neutrophils with intracellular bacteria?
(A) Pure transudate
(B) Septic exudate
(C) Modified transudate
(D) Chylous effusion

2022

204. Which cytological preparation is technically defined as an "imprint" or "touch preparation" from a freshly cut tissue surface?
(A) Fine needle aspirate
(B) Impression smear
(C) Tape preparation
(D) Cytospin

2006

205. Match the anticoagulants in List I with their most appropriate applications in List II:
List I: (Anticoagulant)
P. K3-EDTA
Q. Sodium Citrate
R. Lithium Heparin
S. Sodium Fluoride
List II: (Application)
1. Coagulation profiles (PT/aPTT)
2. Routine mammalian complete blood counts (CBC)
3. Long-term preservation of blood glucose
4. Routine clinical biochemistry and avian hematology
(A) P-2, Q-1, R-4, S-3
(B) P-1, Q-2, R-3, S-4
(C) P-4, Q-3, R-2, S-1
(D) P-2, Q-3, R-1, S-4

NODIA

2002

206. Which is the most appropriate fixative for submitting a whole eye for histopathological examination?
(A) Buffered 10% neutral formalin solutions
(B) Dehydrated 100% methanol cell fixatives
(C) Davidson's fixative or Bouin's solution
(D) Standard sterile physiological saline

2019

207. A dark brown to black coloration of urine that is positive for occult blood on a dipstick but lacks intact red blood cells on sediment examination most likely indicates:
(A) Intact hematuria with RBC sediment
(B) Severe pyuria with cellular casts
(C) High concentration of urobilinogen
(D) Myoglobinuria from rhabdomyolysis

2007

208. Given below are two statements, one labeled as Assertion (A) and the other as **Reason (R):**
Assertion (A): Evaluating a direct smear of fresh feces is essential for diagnosing infections by motile protozoal trophozoites, such as Tritrichomonas foetus.
Reason (R): Standard flotation solutions have

2014

190. Consider the following statements regarding cerebrospinal fluid (CSF) analysis:

I. The fluid should normally clot upon standing.

II. Total protein is normally very low (< 30 mg/dL) compared to serum.

III. The normal nucleated cell count is < 5 cells/ μL, primarily lymphocytes and monocytes.

IV. Erythrophages in the CSF indicate chronic or prior hemorrhage rather than an acute iatrogenic tap.

Which of the above statements is/are INCORRECT?

(A) I only
(B) II and III
(C) I and IV
(D) III and IV

2018

191. The primary purpose of "clearing" a tissue sample during histopathological processing is to:

(A) Remove water from tissues to prevent enzymatic decomposition
(B) Selectively stain cell nuclei for microscopic visualization
(C) Decalcify small pieces of dense bone prior to sectioning
(D) Replace dehydrating alcohol with a wax-miscible solvent

2003

192. Which fixative is specifically recommended when preserving feces for later microscopic examination of protozoan cysts to maintain their delicate morphology?

(A) Standard 10% Neutral Buffered Formalin solutions
(B) Absolute 100% Ethanol dehydration solutions
(C) Polyvinyl Alcohol or Sodium Acetate-Acetic Acid-Formalin
(D) Highly concentrated buffered Glutaraldehyde solutions

2012

193. The specific gravity of standard veterinary sucrose flotation solution utilized for fecal parasite examination is typically adjusted to:

(A) 1.000 to 1.050
(B) 1.100 to 1.150
(C) 1.250 to 1.300
(D) 1.400 to 1.450

2024

194. In feline patients, a severe, life-threatening regenerative anemia caused by a hemotropic mycoplasma attaching to the surface of erythrocytes is known as:

(A) Feline Leukemia Virus infection
(B) Feline Infectious Peritonitis disease
(C) Systemic Cytauxzoonosis infection
(D) Feline Hemotropic Mycoplasmosis

2014

195. Which distinct morphological feature identifies Cytauxzoon felis in the erythrocytes of domestic cats?

(A) Small, round to signet-ring-shaped piroplasms
(B) Large, pairs of pear-shaped bodies
(C) Long, undulating extracellular organisms
(D) Stippled, irregular intracellular inclusion bodies

2011

196. The "Bence Jones protein" detected in urine is actually composed of which specific molecule?

(A) Degraded heavy chains of immunoglobulins
(B) Proteolytic degraded fragments of albumin
(C) Immunoglobulin free light chains
(D) Polymerized Tamm-Horsfall mucoprotein

2007

197. Why do routine veterinary urine dipsticks often fail to reliably detect Bence Jones proteinuria in cases of multiple myeloma?

(A) The reagent pad detects albumin and has very poor sensitivity for light chains.
(B) The evaluated urine sample is typically far too alkaline for chemical reaction.
(C) Bence Jones protein molecules rapidly degrade and evaporate at room temperature.
(D) Active clinical cases of multiple myeloma do not produce any urinary proteins.

2017

198. Which specific erythrocyte index directly calculates the average weight of hemoglobin within a single red blood cell?

(A) Mean Corpuscular Volume (MCV)
(B) Mean Corpuscular Hemoglobin Concentration (MCHC)
(C) Packed Cell Volume (PCV)
(D) Mean Corpuscular Hemoglobin (MCH)

2008

199. What does the presence of significant numbers of nucleated red blood cells (nRBCs) in peripheral

Click Here to Buy book on Amazon

ANSWERS

1. (B) To inhibit bacterial overgrowth while preserving cellular elements
Boric acid is commonly added to veterinary urine specimens to prevent the multiplication of contaminant bacteria without altering the physical properties of the cells or casts. This allows the sample to remain stable during transit when refrigeration is unavailable, ensuring that subsequent cytological evaluations and bacterial cultures yield highly accurate, representative clinical results.

2. (D) Sodium fluoride tube
Sodium fluoride acts as an effective antiglycolytic agent by inhibiting the enolase enzyme in the glycolytic pathway. When blood sample separation is delayed, red blood cells continue to consume glucose in vitro, causing a false decrease. Utilizing sodium fluoride preserves baseline glucose levels during prolonged transit, ensuring precise and reliable biochemical analysis.

3. (A) Echinocytosis and leukocyte pyknosis
Extended storage of whole blood at room temperature alters red cell membrane permeability and cell volume, leading to the formation of crenated cells known as echinocytes. Concurrently, white blood cells undergo degenerative changes, notably nuclear condensation or pyknosis. Immediate smear preparation is critical to avoid these morphological artifacts during microscopic examination.

4. (C) Delayed separation of serum from erythrocytes
Prolonged contact between serum and cellular components allows intracellular potassium to leak from erythrocytes into the serum. This passive leakage occurs even in the absence of visible hemolysis, particularly in certain breeds with high potassium concentrations in their red blood cells. Prompt centrifugation and separation prevent this artificial elevation in serum potassium levels.

5. (A) Chelation of ionized calcium
Ethylenediaminetetraacetic acid prevents coagulation by binding ionized calcium in the sample, making it unavailable for the clotting cascade. Calcium is a critical cofactor in several enzymatic steps of both the intrinsic and extrinsic coagulation pathways. Removing free calcium effectively halts the cascade, maintaining the blood in a fluid state for cellular analysis.

6. (D) Its calcium-binding effect is easily reversible by adding calcium chloride
Sodium citrate acts by binding calcium, but this inhibition is easily reversed in the laboratory by adding calcium chloride back to the plasma. This reversibility is essential for initiating and timing coagulation reactions in assays. Additionally, sodium citrate preserves sensitive coagulation factors, making it the standard anticoagulant for testing clotting function.

7. (B) Lithium heparin
Lithium heparin is the preferred anticoagulant for avian and reptilian blood because ethylenediaminetetraacetic acid often causes significant hemolysis and lysis of nucleated erythrocytes in these species. Heparin prevents clotting by accelerating antithrombin activity, thereby preserving the integrity of fragile red and white blood cells for accurate automated and manual hematological analyses.

8. (C) Cytology slides should be shipped in the same sealed container as formalin-fixed tissues.
Formalin fumes penetrate shipping containers and partially fix cells on cytological smears, which severely impairs their subsequent staining properties. This artifact prevents proper uptake of Romanowsky stains, rendering the cells pale, bluish, and virtually impossible to interpret. Therefore, cytology slides must always be packaged and shipped entirely separate from formalin-fixed tissues.

9. (D) Lipemia
Lipemia introduces significant turbidity to a blood sample, which scatters light during spectrophotometric analysis in automated hematology analyzers. Because hemoglobin is measured by light absorption at a specific wavelength, this increased turbidity is incorrectly read as high optical density, resulting in a falsely

NODIA

2008

152. In diagnosing a coagulation disorder, the buccal mucosal bleeding time (BMBT) is primarily an in vivo test for evaluating:
(A) Factor VIII activity
(B) Secondary hemostasis
(C) Primary hemostasis
(D) Fibrin degradation rate

2001

153. A markedly elevated Total Iron Binding Capacity (TIBC) combined with low serum iron is the classical biochemical pattern for:
(A) Hemolytic anemia
(B) Anemia of chronic disease
(C) Aplastic anemia
(D) Iron deficiency anemia

2014

154. What is the definitive mechanism leading to the formation of schistocytes on a blood smear?
(A) Mechanical shearing by intravascular fibrin strands
(B) Extensive oxidative damage directly to the erythrocyte membrane
(C) Failure of the nucleus to extrude properly during erythropoiesis
(D) Antibody-mediated phagocytosis of the outer erythrocyte membrane

2012

155. Which artifactual error in packed cell volume (PCV) determination occurs when a microhematocrit tube is insufficiently centrifuged?
(A) Falsely decreased PCV due to cellular compression
(B) Falsely elevated PCV due to trapped plasma
(C) Hemolysis resulting in a decreased PCV
(D) Conversion of erythrocytes to echinocytes

2018

156. In the absolute differential leukocyte count, how is the absolute value of a specific cell type calculated?
(A) Total percentage of cell type divided by the Total Leukocyte Count
(B) Fractional percentage of cell type $\times$ Total Leukocyte Count
(C) Total percentage of specific cell type multiplied by one hundred
(D) Total Leukocyte Count divided by the total percentage of cell type

2022

157. A "leukemoid response" in veterinary hematology is defined as:
(A) Extreme non-neoplastic leukocytosis mimicking leukemia
(B) Proliferation of neoplastic white blood cells in peripheral circulation
(C) Sudden rapid destruction of all white blood cell lines by a viral pathogen
(D) Consistent presence of equal proportions of all circulating leukocyte types

2001

158. The presence of Pelger-Huët anomaly in a dog's blood smear is characterized by:
(A) Persistent hypersegmentation observed in all mature neutrophils
(B) Large, giant intracytoplasmic lysosomal granules in leukocytes
(C) Hyposegmentation of granulocyte nuclei with mature chromatin
(D) Sudden, spontaneous erythrocyte autoagglutination in blood smears

2014

159. Which test is utilized to differentiate autoagglutination from simple rouleaux formation in an anemic patient?
(A) Coombs test
(B) Pandy's test
(C) Osmotic fragility test
(D) Saline dispersion test

2014

160. Match the diagnostic tests for gastrointestinal function in List I with their specific diagnostic utilities in List II:

List I: (Diagnostic Test)
P. Serum Cobalamin
Q. Serum Folate
R. TLI (Trypsin-like Immunoreactivity)
S. PLI (Pancreatic Lipase Immunoreactivity)

List II: (Diagnostic Utility)
1. Specific diagnosis of Exocrine Pancreatic Insufficiency
2. Highly specific marker for diagnosing Pancreatitis
3. Absorbed specifically in the proximal small intestine
4. Absorbed specifically in the distal small intestine (ileum)

(A) P-4, Q-3, R-1, S-2
(B) P-3, Q-4, R-2, S-1
(C) P-1, Q-2, R-3, S-4
(D) P-2, Q-1, R-4, S-3

Downloaded from www.pdf.tube

20. (A) P-2, Q-1, R-4, S-3
Acanthocytes exhibit irregular membrane projections, commonly seen in canine hemangiosarcoma and feline hepatic lipidosis. Schistocytes are fragmented red cells indicating physical trauma, characteristic of disseminated intravascular coagulation. Target cells show excess membrane relative to volume, associated with regenerative anemia and liver diseases. Echinocytes feature uniform spikes, which occur in uremic patients or during sample storage.

21. (A) Hemocytometer counting using 1% ammonium oxalate diluent
The manual hemocytometer method remains the reference standard for platelet estimation when automated analyzers fail due to platelet clumping. Diluting the blood with one percent ammonium oxalate lyses mature erythrocytes while preserving platelets and leukocytes. This allows the laboratory technician to count platelets within a grid chamber under a microscope, providing a reliable count.

22. (D) Intrinsic pathway
The activated partial thromboplastin time measures the integrity of both the intrinsic and common coagulation pathways. A normal prothrombin time rules out common pathway abnormalities, as well as extrinsic pathway defects. Therefore, an isolated prolongation of the activated partial thromboplastin time specifically points to a deficiency or inhibition of factors in the intrinsic pathway.

23. (B) Large platelet aggregates at the feathered edge of the smear
Platelet clumping is a common in vitro artifact that falsely lowers the automated platelet count. To verify true thrombocytopenia, the feathered edge of a well-prepared blood smear must be examined microscopically. The presence of large aggregates of platelets at the edge confirms clumping, indicating that platelet production in the patient is likely adequate.

24. (C) Babesia bigemina
Babesia bigemina is a protozoal parasite of ruminants transmitted by ixodid ticks. Under microscopic examination of blood smears, the organism appears inside erythrocytes as large, pear-shaped or piriform trophozoites. These trophozoites characteristically occur in pairs, joined at their narrow ends to form an acute angle, which is a classic diagnostic feature.

25. (D) Monocytes
Ehrlichia canis is a tick-borne intracellular bacterium that specifically targets and infects canine monocytes and macrophages. Inside the host cell cytoplasm, the bacteria replicate and aggregate to form round, basophilic inclusion bodies known as morulae. Finding these morulae on blood smears or buffy coat preparations is diagnostic for canine monocytic ehrlichiosis.

26. (A) Alanine Aminotransferase (ALT)
Alanine aminotransferase is located in the cytoplasm of hepatocytes in small animals. When hepatocyte membranes are damaged or ruptured, this enzyme leaks into the bloodstream, making it a highly sensitive and specific biomarker for active liver injury. Because of its minimal presence in other tissues, elevated levels directly indicate hepatocellular damage.

27. (B) Sorbitol Dehydrogenase (SDH)
Large animals have negligible alanine aminotransferase activity in their hepatocytes, rendering it useless for diagnosing liver disease. Instead, sorbitol dehydrogenase is highly specific and sensitive for detecting acute hepatocellular necrosis in ruminants and horses. Because of its short half-life in circulation, elevated levels indicate active, ongoing liver cell damage in these species.

28. (B) Cholestasis
Alkaline phosphatase and gamma-glutamyl transferase are membrane-bound enzymes associated with the biliary canalicular membrane. Impedance of bile flow, or cholestasis, stimulates increased synthesis and release of these enzymes into blood. When this occurs without a significant rise in cytoplasmic alanine aminotransferase, it indicates a primarily cholestatic or obstructive process rather than direct hepatocellular injury.

29. (A) Serum albumin
Albumin is synthesized exclusively by hepatocytes in the liver. While liver enzymes indicate active cell damage or biliary disease, they do not reflect how well the liver is functioning. A decline in serum albumin, in the absence of renal or gastrointestinal loss, indicates a significant loss of the liver's synthetic capacity.

NODIA

differentiate neoplastic cells from normal epithelial tissue or benign hyperplasia.

50. (B) I, II and V
Urine specific gravity measures the concentration of dissolved solutes, reflecting the kidney's ability to concentrate or dilute urine. Isosthenuria occurs when urine specific gravity matches glomerular filtrate, between 1.008 and 1.012. A value of 1.045 in a dehydrated dog shows healthy tubular function, whereas refractometers are far more accurate than clinical dipsticks.

51. (A) Altered carbohydrate and lipid metabolism
Ketonuria occurs when cells cannot utilize glucose for energy, forcing the body to mobilize lipids for fuel. This rapid lipid metabolism leads to the production of acetoacetate, beta-hydroxybutyrate, and acetone in the liver. These ketones filter through the glomerulus and enter the urine, commonly signaling diabetic ketoacidosis or starvation in dogs.

52. (C) Cystine
Cystine crystals are flat, colorless, six-sided hexagons found in acidic urine. Their presence is a pathognomonic indicator of cystinuria, an inherited metabolic defect of renal tubular reabsorption affecting cystine and other amino acids. Because they are not found in normal urine, finding these crystals is diagnostic for this specific genetic disorder.

53. (A) Renal tubules
Urinary casts are cylindrical structures formed in the lumen of the distal renal tubules and collecting ducts. Cellular casts contain sloughed epithelial cells, indicating acute tubular injury, degeneration, or necrosis. The presence of these casts on urine sediment examination is a highly specific indicator of active parenchymal disease localized within the renal tubules.

54. (D) Hemoglobin
Fecal occult blood tests utilize chemical reagents that detect the presence of heme. The hemoglobin molecule contains a heme group that possesses pseudoperoxidase activity. This catalytic activity facilitates the oxidation of a chromogen indicator by hydrogen peroxide, producing a visible color change that confirms the presence of microscopic bleeding in the gastrointestinal tract.

55. (B) Zinc sulfate solution
Zinc sulfate is the preferred flotation solution for identifying Giardia cysts because its specific gravity is optimized to float the fragile cysts while minimizing osmotic distortion. Other highly concentrated salt or sugar solutions cause rapid osmotic dehydration, shriveling the cysts and making them difficult to recognize under a microscope during routine fecal analysis.

56. (C) Demodex canis
Demodex canis is a microscopic mite that resides deep within the hair follicles and sebaceous glands of the skin. To retrieve these mites for diagnostic identification, the skin must be squeezed and scraped deeply enough to rupture the follicles and capillary beds, ensuring that blood and follicular debris are present on the slide.

57. (D) Lactophenol cotton blue
Lactophenol cotton blue is the standard stain used to evaluate hair shafts and fungal cultures. Phenol kills any live organisms, lactic acid preserves the fungal structures, and cotton blue stains the chitin in the fungal cell walls. This highlights hyphae and macroconidia, allowing for clear morphological identification under a light microscope.

58. (A) Globulins
Pandy's test is a rapid qualitative method used to evaluate cerebrospinal fluid for elevated protein concentrations. The reagent, containing saturated phenol, selectively precipitates globulins, producing a turbid or cloudy appearance. This allows clinicians to quickly detect a localized or systemic immune response, as normal cerebrospinal fluid contains virtually no globulins.

59. (B) Suppurative pleocytosis
Pleocytosis describes an abnormal increase in the nucleated cell count of cerebrospinal fluid. When neutrophils are the dominant cell type present, the condition is termed suppurative pleocytosis. This cytological pattern is strongly associated with bacterial meningitis, fungal infections, or severe non-infectious inflammatory disorders of the central nervous system.

60. (B) Fixation, Dehydration, Clearing, Embedding, Sectioning
Histopathological preparation begins with tissue fixation to preserve structure. Next, dehydration

Click Here to Buy book on Flipkart

40. (C) II and III
Fibrinogen is consumed during clotting, meaning it is absent in serum but present in plasma. Therefore, total protein measurements on serum exclude fibrinogen. Additionally, positive acute-phase proteins like C-reactive protein increase significantly during acute inflammation. Thus, the statements claiming fibrinogen is measured in serum and that C-reactive protein decreases are incorrect.

41. (D) Chronic antigenic stimulation
Chronic antigenic stimulation, such as from persistent infectious diseases or chronic inflammatory conditions, triggers the immune system to produce large quantities of immunoglobulins. This absolute increase in globulin levels alters the proportion of serum proteins, leading to a marked decrease in the calculated albumin-to-globulin ratio, despite albumin concentrations remaining stable or slightly decreased.

42. (A) Hyperventilation resulting in a decreased PCO_2
Metabolic acidosis reduces blood pH, stimulating peripheral and central chemoreceptors to increase the rate and depth of respiration. This hyperventilation causes rapid elimination of carbon dioxide, which decreases the partial pressure of carbon dioxide in arterial blood. This respiratory compensation helps return the blood pH toward the normal physiological reference range.

43. (B) Collection in an EDTA tube
Ethylenediaminetetraacetic acid prevents coagulation by strongly chelating divalent cations, particularly calcium, making them unavailable. If a blood sample intended for biochemistry is collected in an EDTA tube, or contaminated with EDTA from another tube, the measured serum calcium levels will be extremely and falsely low, often approaching zero.

44. (B) Potassium
Potassium is the primary intracellular cation, maintained at high concentrations inside cells by the sodium-potassium ATPase pump. This concentration gradient across the cell membrane is fundamental for establishing and maintaining the resting membrane potential of excitable tissues. Evaluating serum potassium levels is vital, as small changes can severely affect cardiac conduction.

45. (A) They excellently highlight cytoplasmic details and microorganisms.
Romanowsky-type stains are ideal for veterinary clinical cytology because they provide excellent differentiation of cytoplasmic details, granules, and infectious organisms. These stains consist of acidic and basic dyes that color cell components characteristically. This allows for quick and detailed examination of inflammatory cells, neoplastic cells, and extracellular matrix in the clinic.

46. (C) Squash (compression) preparation
The squash preparation technique is ideal for semi-solid, highly cellular aspirates. A second slide is placed flat onto the sample drop and gently slid across the primary slide without applying excessive downward pressure. This spreads the cells into a monolayer, allowing proper staining and preventing the thick clumps that hinder microscopic evaluation.

47. (D) Total protein < 2.5 g/dL and Total Nucleated Cell Count $< 1{,}500/\mu L$
Pure transudates are non-inflammatory fluids that accumulate in body cavities due to systemic imbalances in hydrostatic or oncotic pressure. They are characterized by a low protein content, typically less than two and a half grams per deciliter, and a low total nucleated cell count, usually below fifteen hundred cells per microliter.

48. (C) Suppurative (purulent)
Suppurative or purulent inflammation is characterized by a high proportion of neutrophils, typically exceeding eighty percent of the total nucleated cell population. The presence of degenerate neutrophils indicates cell swelling and lysis, usually caused by bacterial toxins. The remaining fraction is composed of macrophages, which act to phagocytize debris at the lesion site.

49. (B) Macronucleoli and multinucleation
A diagnostic hallmark of malignancy in cytological specimens is the presence of nucleolar abnormalities, such as macronucleoli, and multinucleation within a single cell. These features reflect abnormal cell division and highly active protein synthesis characteristic of malignant tumors, helping cytologists

Downloaded from www.pdf.tube

gastrointestinal loss of bicarbonate ions from the body.

112. (C) Collecting naturally exfoliated or scraped cells
Exfoliative cytology involves the collection and microscopic evaluation of cells that are shed naturally from epithelial surfaces or harvested through gentle mechanical scraping. This technique provides immediate diagnostic information about superficial lesions, mucosal linings, and bodily fluids without requiring invasive surgical procedures or core tissue biopsy.

113. (B) Normal or reactive lymph node
A cytological specimen containing more than eighty percent small, mature lymphocytes with a minor population of larger immunoblasts, macrophages, and plasma cells is characteristic of normal or hyperplastic lymph tissue. This heterogenous population indicates an active immune response rather than the monomorphic cell population typically associated with lymphoid neoplasia.

114. (D) A viscous, high-protein exudate with non-degenerate neutrophils
Feline infectious peritonitis classically produces a sterile, clear-to-straw-colored peritoneal or pleural effusion with a high protein concentration and relatively low cellularity. Cytological evaluation reveals a background of abundant proteinaceous precipitate, paired with a mixed population of non-degenerate neutrophils, macrophages, and plasma cells, consistent with immune-mediated vasculitis.

115. (A) Round cells containing distinct purple intracytoplasmic granules
Mast cell tumors are classified as discrete round cell neoplasms. Cytological evaluation of these masses typically yields highly cellular aspirates consisting of individual round cells characterized by abundant, metachromatic, purple intracytoplasmic granules. These granules contain histamine, heparin, and other inflammatory mediators, which stain intensely with standard Romanowsky-type stains.

116. (C) Acidic (pH 5.5 to 7.0)
Diet is the primary physiological determinant of urine pH in domestic animals. Diets rich in animal protein generate a higher load of acidic metabolic products, such as sulfuric and phosphoric acids, which are subsequently excreted by the kidneys. This process results in the production of consistently acidic urine in healthy dogs and cats.

NODIA

117. (A) Glucose
Reagent strips designed for urinalysis detect urinary sugar using a highly specific double sequential enzymatic reaction. The enzyme glucose oxidase reacts specifically with glucose to produce gluconic acid and hydrogen peroxide. This specific reaction ensures that other sugars, including lactose, fructose, and galactose, do not cause false-positive results on the testing pad.

118. (D) Hyaline cast
Hyaline casts are formed through the precipitation of Tamm-Horsfall mucoprotein within the lumen of the renal tubules. These structures are colorless, semi-transparent, and devoid of any cellular inclusions. While they can be observed in low numbers in healthy animals, increased numbers indicate mild glomerular proteinuria or renal congestion.

119. (B) Trichuris vulpis
Trichuris vulpis is the common whipworm infecting dogs. The ova are easily identified during microscopic fecal examination due to their characteristic symmetrical, football-like shape, thick yellow-brown shell, and prominent bipolar plugs. These distinct features differentiate them from hookworm or roundworm eggs, which lack these bipolar structures entirely.

120. (B) II only
For appropriate histopathological preservation, tissue samples must be placed in ten percent neutral buffered formalin at a minimum volume ratio of ten parts fixative to one part tissue. A one-to-one ratio is completely inadequate, leading to poor fixative penetration, delayed preservation, and subsequent autolysis of the specimen.

121. (D) Sudan III or IV
Sudan III and Sudan IV are lipophilic dyes that bind specifically to neutral lipids and triglycerides. Applying these stains to a fecal smear allows direct microscopic visualization of undigested fat droplets, which appear as bright orange-red spheres. This quick screening method helps confirm steatorrhea associated with exocrine pancreatic insufficiency.

removes water using graded alcohols, followed by clearing with a solvent to prepare the tissue for paraffin. The sample is then embedded in a paraffin block, which provides rigid support, and finally sliced into ultra-thin slices during the sectioning stage.

61. (A) To ensure complete, uniform tissue penetration
An adequate volume of fixative is critical to prevent tissue autolysis and decay. A ratio of ten parts formalin to one part tissue ensures there is sufficient formaldehyde available to completely penetrate the tissue sample and cross-link proteins throughout the entire specimen, preserving the cellular architecture uniformly for subsequent processing and staining.

62. (C) Jugular vein
The jugular vein is the preferred site for collecting large volumes of blood in adult cattle. It provides a large, easily accessible vessel that minimizes turbulence during draw, reducing the risk of hemolysis. This site ensures rapid collection of high-quality samples, which is essential for accurate clinical pathology and biochemical testing.

63. (D) Falsely decreased due to cellular glycolysis
Red blood cells and leukocytes continue to consume glucose via glycolysis while in contact with serum. If the serum is not separated promptly from the cellular clot, glucose levels decrease artificially at a rate of approximately seven to ten percent per hour. This pre-analytical error can lead to a false diagnosis of hypoglycemia.

64. (C) Relative polycythemia
Relative polycythemia occurs when a decrease in plasma volume concentrates the cellular components of blood. In a severely dehydrated animal, the total erythrocyte count, packed cell volume, and hemoglobin are elevated, but the absolute mass of red blood cells remains normal. Rehydration restores normal plasma volume, returning these values to baseline.

65. (B) Splenic dysfunction or splenectomy
Howell-Jolly bodies are small, round, basophilic nuclear remnants left behind during erythrocyte maturation. Normally, the spleen efficiently removes these abnormal cells from circulation via pitting. In animals with splenic dysfunction or those that have undergone a splenectomy, these inclusions persist in circulation, making them a common finding on blood films.

66. (D) Neutrophilia, lymphopenia, and eosinopenia
Corticosteroids, released during stress or exogenous administration, alter leukocyte kinetics. This results in a classic stress leukogram, characterized by neutrophilia due to reduced margination, lymphopenia from tissue sequestration, and eosinopenia. Monocytosis may also occur, particularly in dogs. Identifying this pattern helps clinicians differentiate stress from active systemic inflammation.

67. (A) Impedance technology
Impedance technology counts and sizes cells based on changes in electrical resistance. Blood cells are suspended in an electrolyte solution and drawn through a small aperture. Because cells are poor conductors, each passing cell interrupts the electrical current. The number of pulses corresponds to the cell count, while pulse height reflects volume.

68. (C) Cytoplasmic vacuolation and Döhle bodies
Toxic change in neutrophils indicates accelerated production and shortened maturation time in the bone marrow, usually driven by severe inflammation. Cytoplasmic vacuolation, Döhle bodies, diffuse cytoplasmic basophilia, and toxic granulation are the classic morphological abnormalities observed. Recognizing these features on a blood smear helps confirm the presence of systemic inflammation.

69. (A) Inside erythrocytes
Babesia gibsoni is a small, intraerythrocytic piroplasm that infects dogs, causing hemolytic anemia. Once inside the host, the sporozoites invade red blood cells, where they replicate via binary fission, forming merozoites. This intraerythrocytic replication leads to mechanical damage of the cell membrane, immune-mediated destruction, and clinical signs of hemolysis.

70. (A) II, IV, I, III
Erythropoiesis begins with the rubriblast, the most immature committed erythroid precursor. It differentiates into the prorubricyte, followed by the rubricyte as chromatin condenses. The nucleus is then extruded at the metarubricyte stage, transitioning into an enucleated reticulocyte. This chronological sequence is

Click Here to Buy book on Amazon

essential for evaluating bone marrow response to anemia.

71. (B) Hepatocyte mitochondria
Glutamate dehydrogenase is a mitochondrial enzyme found in high concentrations in the liver of ruminants. Because it is localized within the mitochondria rather than the cytoplasm, elevated serum GLDH levels are highly specific indicators of severe, necrotizing hepatocellular damage, making it a valuable biomarker for evaluating liver disease in cattle and sheep.

72. (C) Osteoblastic activity in growing bones
Alkaline phosphatase exists in several tissue-specific isoforms, including hepatic and bone-specific forms. In young, rapidly growing puppies, active bone remodeling and osteoblastic activity release high amounts of the bone ALP isoform into circulation. This physiological process causes a normal elevation in total serum ALP, which should not be misinterpreted as cholestasis.

73. (D) Conjugated bilirubin
Unconjugated bilirubin, bound to albumin, is taken up by hepatocytes and conjugated with glucuronic acid. This conjugation process converts the lipid-soluble pigment into water-soluble conjugated bilirubin. Once water-soluble, it is actively transported across the canalicular membrane into the bile duct system for eventual excretion into the gastrointestinal tract.

74. (A) Severe hepatic insufficiency
Urea is synthesized exclusively by the liver from ammonia via the urea cycle. In cases of severe hepatic insufficiency or portosystemic shunts, the liver cannot efficiently convert ammonia into urea. This loss of synthetic function leads to a significant decrease in blood urea nitrogen levels, accompanied by an elevation in blood ammonia.

75. (B) Increased vascular permeability
Exudates are inflammatory fluids that accumulate when inflammatory mediators increase vascular permeability. This allows plasma proteins and nucleated cells to escape from capillaries into the pleural or peritoneal space. Consequently, exudative effusions are characterized by high protein concentrations and high cellularity, helping to distinguish them from transudative fluids.

76. (B) Hemoglobinuria or myoglobinuria
The chemical reagent on urine dipsticks reacts with heme, producing a positive result for blood. If microscopic examination of the sediment reveals no intact red blood cells, the positive reaction is caused by free hemoglobin or myoglobin in the urine. This indicates intravascular hemolysis or severe muscle necrosis, respectively.

77. (A) Waxy cast
Waxy casts represent the final stage of cellular and granular cast degradation within the renal tubules. Their presence indicates chronic, low-flow tubular passage, allowing the proteinaceous material to become highly condensed and brittle. Thus, finding waxy casts is a significant indicator of localized nephron obstruction and chronic renal disease.

NODIA

78. (C) Nematode larvae
The Baermann technique is a specialized parasitological method designed to isolate live nematode larvae from fecal samples. It relies on the active migration of larvae out of feces into warm water, where they sink to the bottom of the apparatus. This technique is essential for diagnosing lungworm infections, such as Aelurostrongylus.

79. (D) Globulin
The Nonne-Apelt test is a classic semi-quantitative assay used to detect increased globulin levels in cerebrospinal fluid. The reagent, consisting of saturated ammonium sulfate, selectively precipitates globulins, forming a white ring or cloudiness. This clinical test helps identify localized immunoglobulin production associated with central nervous system inflammation or infection.

80. (A) Both A and R are true and R is the correct explanation of A.
Ethylene glycol ingestion is a veterinary emergency. Ingested glycol is metabolized into oxalic acid, which chelates systemic calcium to form calcium oxalate. These crystals precipitate in the renal tubules, causing acute tubular necrosis. The presence of calcium oxalate monohydrate crystals in urine sediment confirms this pathological process, supporting the clinical diagnosis.

81. (B) Hepatic fibrosis
Fine needle aspiration retrieves individual cells, which is excellent for cytological evaluation of diffuse diseases like lipidosis or lymphoma. However, diagnosing hepatic fibrosis requires assessing tissue architecture and the distribution of extracellular matrix. A core biopsy preserves the tissue structure, allowing histopathologists to visualize and grade connective tissue deposition.

82. (D) Precipitated ribosomal RNA
New methylene blue is a supravital stain that enters living, unfixed erythrocytes. It binds to residual ribosomal ribonucleic acid and organelles within immature red blood cells, causing them to precipitate into visible, dark blue networks or granules. This allows for the precise identification and quantification of reticulocytes to assess regenerative capacity.

83. (A) Elevated BUN and creatinine
Azotemia refers to the accumulation of non-protein nitrogenous waste products in the blood, specifically blood urea nitrogen and creatinine. It is defined strictly by these biochemistry findings, regardless of the underlying cause. Azotemia is categorized into pre-renal, primary renal, or post-renal forms based on urine specific gravity and clinical findings.

84. (C) Albumin
During acute inflammation, cytokines prompt the liver to prioritize the synthesis of positive acute-phase proteins involved in host defense. Consequently, the production of other proteins, specifically albumin, is down-regulated. This makes albumin a classic negative acute-phase protein, and its serum levels drop during active systemic inflammatory processes.

85. (A) Altered concentrations of specific serum proteins
Dysproteinemia refers to any abnormal alteration in the concentrations, types, or relative proportions of proteins in the blood. This includes scenarios like monoclonal gammopathies, polyclonal hyperglobulinemia, or severe hypoalbuminemia. Identifying these dysproportions through serum protein electrophoresis is critical for diagnosing immune-mediated, infectious, or neoplastic diseases.

86. (D) Hyperkalemia
Urethral obstruction or bladder rupture impairs the excretion of potassium, which is normally eliminated via urine. Because potassium cannot leave the body, it accumulates in extracellular fluid, causing life-threatening hyperkalemia. This electrolyte imbalance alters myocardial cell resting membrane potential, leading to bradycardia and potentially fatal cardiac arrhythmias.

87. (B) Chronic increases in hydrostatic pressure with protein addition
Modified transudates are transitional fluids that form when increased hydrostatic pressure or lymphatic obstruction forces protein and cells into a body cavity. This often occurs with congestive heart failure or portal hypertension. These fluids have higher protein and cellularity than pure transudates but lower levels than inflammatory exudates.

88. (C) Uniform population of cells with a low $N{:}C$ ratio
Benign neoplasms are characterized by slow, controlled cell growth, resulting in a uniform population of well-differentiated cells. These cells closely resemble their tissue of origin, showing minimal variation in nuclear size and maintaining a low nucleus-to-cytoplasm ratio. The absence of aggressive cellular criteria suggests a non-invasive, benign process.

89. (D) Leaving the sample unsealed at room temperature
Leaving a urine sample unsealed at room temperature allows dissolved carbon dioxide to escape into the air. This loss of carbon dioxide shifts the chemical equilibrium, raising the pH. Additionally, contaminant urease-producing bacteria can multiply and convert urea into ammonia, further inflating the measured urine pH falsely.

90. (A) P-2, Q-3, R-4, S-1
Ammonium biurate crystals appear as brown spheres with spicules, resembling a classic thorn apple shape. Calcium oxalate dihydrate crystals typically form small, square envelopes containing an internal cross. Bilirubin crystals present as golden-brown, needle-like bundles in acidic urine. Cystine crystals are characterized by colorless, hexagonal plates, signaling an inherited metabolic defect.

Downloaded from www.pdf.tube

30. (A) I and II
Bile acids are synthesized from cholesterol in hepatocytes and secreted into bile. Measuring fasting and post-prandial concentrations assesses the hepatic clearance of portal blood, serving as a highly sensitive test of overall liver function. However, elevated concentrations are non-specific indicators of hepatic dysfunction or portosystemic shunts, not a specific diagnostic marker for cholangiocarcinoma.

31. (D) Extravascular hemolytic anemia
Extravascular hemolysis results in excessive breakdown of hemoglobin by macrophages, releasing large amounts of unconjugated bilirubin into circulation. This high influx overwhelms the liver's capacity to conjugate the pigment. Consequently, unconjugated bilirubin increases in the serum, while conjugated bilirubin remains normal because the excretory pathways of the hepatobiliary system are not obstructed.

32. (C) Kidney
Symmetric dimethylarginine is excreted almost exclusively by renal filtration. It serves as a highly sensitive biomarker for kidney function, increasing with as little as a twenty-five percent reduction in glomerular filtration rate. This allows for the early detection of chronic kidney disease in dogs and cats, long before traditional markers like creatinine rise.

33. (B) BUN is influenced by dietary protein and gastrointestinal bleeding.
Blood urea nitrogen is highly susceptible to non-renal influences, such as dietary protein content, gastrointestinal hemorrhage, and protein catabolism, which can cause significant fluctuations. In contrast, serum creatinine is produced at a relatively constant rate from muscle metabolism and is minimally affected by diet. This stability makes creatinine a more specific indicator of glomerular filtration.

34. (D) Uric acid
Birds and reptiles are uricotelic, meaning they excrete nitrogenous waste primarily as uric acid rather than urea. Uric acid is synthesized in the liver and actively secreted by the renal tubules. Therefore, evaluating blood uric acid concentrations is the standard and most reliable method to assess renal function and detect gout in these animal classes.

35. (A) Urine specific gravity
Pre-renal azotemia results from hypovolemia or decreased renal perfusion, prompting healthy kidneys to conserve water and produce highly concentrated urine. In contrast, primary renal azotemia involves damaged tubules that cannot concentrate urine effectively, leading to isosthenuria. Evaluating urine specific gravity alongside azotemia allows clinical differentiation between pre-renal and primary renal failure.

36. (C) Trypsin-like immunoreactivity (TLI)
The canine serum trypsin-like immunoreactivity test measures both trypsinogen and trypsin circulating in blood. Because trypsinogen is produced exclusively by the exocrine pancreas, a severe reduction in its concentration is highly specific and sensitive for pancreatic acinar atrophy. This makes it the diagnostic gold standard for exocrine pancreatic insufficiency in canine patients.

37. (A) Small intestinal bacterial overgrowth (SIBO)
Small intestinal bacterial overgrowth features an abnormal proliferation of bacteria that consume dietary cobalamin while simultaneously synthesizing folate in the intestinal lumen. Consequently, the host absorbs less cobalamin and more folate, resulting in the classic serum biochemical pattern of hypocobalaminemia and hyperfolatemia, which is highly diagnostic for this specific malabsorptive condition.

NODIA

38. (D) Cardiac Troponin I (cTnI)
Cardiac troponin I is a structural protein unique to the myocardium, regulating cardiac muscle contraction. When myocardial cell damage occurs due to trauma, ischemia, or myocarditis, this protein is rapidly released into the blood. Measuring its concentration provides high diagnostic specificity and sensitivity for detecting acute and chronic myocardial injury in veterinary patients.

39. (B) Creatine Kinase (CK)
Aspartate aminotransferase is located in both skeletal muscle cells and hepatocytes, so elevations can indicate either muscle damage or liver disease. Creatine kinase, however, is highly specific to skeletal muscle tissue. Evaluating both enzymes concurrently allows clinical differentiation; a normal creatine kinase with elevated aspartate aminotransferase points directly to a primary hepatic disorder.

increased consumption during active clotting, sequestration in the spleen, or immune-mediated destruction. Identifying thrombocytopenia is critical, as severe cases can lead to spontaneous hemorrhage.

102. (D) At the periphery of the cell
Anaplasma marginale is an intracellular rickettsial pathogen that infects ruminant erythrocytes. Under light microscopy of a stained blood film, the organism appears as a small, round, dark-purple inclusion. It is characteristically situated at the extreme margin or periphery of the red blood cell, which distinguishes it from other intraerythrocytic inclusions.

103. (B) ALP
Alkaline phosphatase is a membrane-bound enzyme localized to the canalicular membrane of hepatocytes and biliary epithelial cells. Under conditions of impaired bile flow, or cholestasis, bile acids accumulate and stimulate increased synthesis and release of this enzyme into blood. Elevated ALP is therefore a primary diagnostic marker for cholestatic disease.

104. (C) Gallbladder contraction releases bile acids, which are then cleared from portal blood by a healthy liver.
In healthy animals, pre-prandial bile acids are low because they are stored in the gallbladder. Ingesting food triggers cholecystokinin release, causing gallbladder contraction and bile excretion into the duodenum. Reabsorbed bile acids enter the portal circulation and are cleared by hepatocytes. Elevated post-prandial bile acids indicate impaired hepatic uptake or vascular shunting.

105. (D) Hypothyroidism
Thyroid hormones play a crucial role in lipid metabolism, particularly by stimulating the expression of low-density lipoprotein receptors in hepatocytes. In hypothyroid dogs, the lack of thyroid hormones impairs hepatic clearance of cholesterol from circulation. This metabolic disruption leads to marked hypercholesterolemia, making it a common biochemistry finding in hypothyroidism.

106. (A) 25%
Symmetric dimethylarginine serves as a sensitive biomarker for detecting early renal functional decline. It begins to rise when approximately twenty-five percent of nephron function is lost, whereas traditional serum creatinine concentrations typically remain within reference limits until at least seventy-five percent of functional nephron mass has been severely compromised.

107. (B) Urethral obstruction
Post-renal azotemia arises from physical blockages or disruptions in the urinary tract downstream from the kidneys, which prevent normal urine excretion. Blockage of the urethra stops urine flow, causing backpressure that reduces glomerular filtration rate and leads to systemic accumulation of nitrogenous waste products like blood urea nitrogen and creatinine.

108. (B) Glycemic control in diabetes mellitus
Serum fructosamine is formed through a non-enzymatic reaction linking glucose to albumin and other plasma proteins. Because circulating proteins have a half-life of one to two weeks in dogs and cats, measuring fructosamine provides a reliable indicator of average blood glucose concentrations over the preceding fortnight, reflecting overall diabetic control.

109. (A) Increased Total Protein, Increased PCV
Severe acute dehydration leads to a contraction of the intravascular fluid volume, known as hemoconcentration. Because the liquid portion of the blood decreases while the cellular elements and large proteins remain constant, both the packed cell volume and total protein concentration show a relative, artifactual increase on diagnostic testing.

110. (B) II only
Hemolysis releases intracellular potassium into the serum, causing artifactual hyperkalemia in species or breeds with high-potassium erythrocytes, such as horses, pigs, and certain Asian dog breeds. However, most domestic dogs and cats have low-potassium red blood cells, meaning hemolysis does not cause a significant artifactual increase in these species.

111. (D) Metabolic acidosis
Metabolic acidosis is characterized by a primary decrease in blood bicarbonate concentration alongside a reduction in systemic pH. This systemic acid-base disturbance occurs due to either excessive accumulation of organic acids, such as lactate or ketones, or excessive renal or

Click Here to Buy book on Flipkart

91. (B) Suspended particulate matter
Flocculent urine contains large, visible, suspended particulate matter or flakes that settle out of suspension upon standing. This macroscopic appearance is distinct from uniform turbidity and usually indicates the presence of cells, mucus, fibrin, or casts. Evaluating this physical property is an important first step in urine analysis before microscopic sedimentation.

92. (B) Glutaraldehyde
Glutaraldehyde is the preferred fixative for electron microscopy because it contains two aldehyde groups, allowing for superior cross-linking of proteins. This provides exceptional preservation of ultra-structural details and organelles, which is required for high-magnification electron imaging. In contrast, formaldehyde-based fixatives are better suited for standard light microscopic evaluation.

93. (A) Provides a larger sample showing the margin between normal and abnormal tissue
A wedge biopsy involves surgically removing a pie-shaped tissue segment, providing a large, intact specimen. This allows histopathologists to evaluate the critical transition zone between normal tissue and the lesion margin. It provides superior diagnostic information compared to punch biopsies, which are limited by sample size and depth of penetration.

94. (C) Leukopenia
Leukopenia is the clinical term defining a decrease in the total white blood cell count below the established reference interval. This reduction can involve specific leukocyte lines, such as neutrophils or lymphocytes, and usually arises from decreased bone marrow production, increased tissue consumption, or peripheral destruction during severe disease.

95. (D) Plasma contains fibrinogen, while serum does not have fibrinogen.
The critical difference between plasma and serum is the presence of fibrinogen. Plasma is obtained by centrifuging unclotted blood collected with an anticoagulant, thereby retaining fibrinogen. Serum is harvested after blood is allowed to clot naturally, during which fibrinogen is converted into fibrin and incorporated into the clot, leaving the serum fluid.

NODIA

96. (C) Lipemia
Fasting an animal for twelve hours ensures that dietary lipids, particularly chylomicrons, are cleared from circulation. Post-prandial blood draws often display marked lipemia, which causes significant turbidity in the sample. This turbidity interferes with spectrophotometric assays in biochemistry analyzers, leading to false results for various serum components.

97. (B) Marked erythroid regeneration
Macrocytic, hypochromic erythrocyte indices indicate that the bone marrow is actively responding to anemia by releasing immature red blood cells, specifically reticulocytes. These cells are larger than mature erythrocytes, which increases cell volume, and contain less hemoglobin, which lowers cell hemoglobin concentration. This classic pattern confirms a regenerative anemia.

98. (D) Reticulocyte
Polychromatophils appear on Romanowsky-stained blood smears as large red blood cells with a bluish-gray tint, reflecting residual ribosomal RNA. When the same blood sample is stained with the supravital dye new methylene blue, this RNA precipitates into blue granules or networks, classifying these cells as reticulocytes.

99. (A) Liver disease
Target cells, or codocytes, have an excess of cell membrane relative to intracellular hemoglobin, causing a central area of pigment surrounded by a clear ring. This morphology commonly occurs in dogs with liver disease due to alterations in blood lipid concentrations, which increases cholesterol loading in the erythrocyte membrane.

100. (B) I and III
Eosinophilia is a hallmark of type one hypersensitivity reactions and parasite infestations, as mast cells and parasites attract eosinophils. Additionally, lymphopenia is a consistent feature of a corticosteroid-induced stress leukogram. However, monocytosis is not exclusive to viral infections, as it occurs in chronic inflammation, tissue necrosis, and stress.

101. (A) Thrombocytopenia
Thrombocytopenia is the clinical term defining a circulating platelet count below the normal reference interval. This condition can result from decreased bone marrow production,

122. (A) Cheyletiella spp.
Cheyletiella species are highly contagious surface-dwelling mites that infect dogs, cats, and rabbits. These mites live within the keratin layer of the skin and move rapidly along the hair shafts, carrying loose dander on their bodies. This movement creates the classic clinical appearance commonly referred to as walking dandruff.

123. (B) Colorless and clear
Normal cerebrospinal fluid in domestic animals is an ultrafiltrate of plasma that is entirely clear, colorless, and completely free of turbidity. The absolute absence of color and turbidity indicates extremely low protein concentrations and a minimal cellular population, which are key physiological parameters of a healthy central nervous system.

124. (B) A yellowish discoloration from prior hemorrhage
Xanthochromia refers to a yellow discoloration of cerebrospinal fluid resulting from the breakdown of hemoglobin into bilirubin following prior subarachnoid hemorrhage. This finding helps differentiate authentic pathological bleeding, which occurred hours to days before collection, from fresh, pinkish iatrogenic blood contamination introduced during the puncture procedure itself.

125. (A) Fine needle aspiration or biopsy
Differentiating between inflammatory skin lesions and neoplastic skin masses requires direct microscopic evaluation of the cellular components. Fine needle aspiration or tissue biopsy provides the necessary cellular or histopathological details to observe inflammatory cells, tissue architecture, or malignant cellular criteria, which cannot be determined via blood work or urinalysis.

126. (C) 4.5 to 5.5 mL
Blood consists of cellular elements and fluid plasma. A canine blood sample with a packed cell volume of forty-five percent contains forty-five percent cells and fifty-five percent fluid. Consequently, centrifuging a ten-milliliter sample will yield approximately five and a half milliliters of serum or plasma after complete clotting is achieved.

127. (D) It causes severe hypocalcemia and hyperkalemia
Ethylenediaminetetraacetic acid acts as an anticoagulant by chelating divalent cations, particularly calcium and magnesium, which are essential for the coagulation cascade. Testing an EDTA-anticoagulated sample on a biochemistry analyzer results in artifactual, life-threateningly low calcium measurements alongside falsely, extremely elevated potassium values due to the potassium salt additive.

128. (C) Macrocytic, normochromic anemia
Mean corpuscular volume measures the average size of red blood cells, while mean corpuscular hemoglobin concentration reflects the average concentration of hemoglobin inside them. An elevated volume indicates larger-than-normal erythrocytes, while a normal hemoglobin concentration indicates standard hemoglobin density, collectively defining a macrocytic, normochromic anemia pattern.

129. (B) Horse
Rouleaux formation is the physiological stacking of red blood cells in a flat, coin-like arrangement. This occurs due to elevated plasma protein concentrations, which reduce the natural repulsive electrical charges on erythrocyte surfaces. While abnormal in dogs and cats, prominent rouleaux formation is a normal finding in healthy equine blood.

130. (A) I, III, II, IV
Preparing a standard blood smear begins by placing a droplet of blood near the frosted end of a clean slide. Next, a spreader slide drawn back at an angle of thirty to forty-five degrees is pushed forward to create a thin monolayer. The slide is rapidly air-dried and then stained.

NODIA

131. (A) Eosinophil
Eosinophils are granulocytes characterized by prominent, round, reddish-orange cytoplasmic granules in most domestic species. These cells play a critical role in the immune system by defending the host against helminthic parasitic infections and modulating immediate hypersensitivity and allergic reactions through the release of specialized granular enzymes.

insufficiency, or severe gastrointestinal malabsorption, and is characterized by voluminous, greasy, pale, or foul-smelling stools containing numerous microscopic lipid droplets on a fecal smear.

173. (A) Black, tarry appearance (Melena)
Melena is characterized by dark, black, tarry feces resulting from the digestion of blood within the upper gastrointestinal tract. As erythrocytes pass through the stomach and small intestine, gastric acid and digestive enzymes digest the hemoglobin, turning it black before it is excreted, indicating bleeding from the stomach or duodenum.

174. (C) Dermatophyte Test Medium (DTM)
Dermatophyte Test Medium is a specialized selective agar used to diagnose fungal infections of the skin, hair, and nails. DTM contains phytone, dextrose, and phenol red, which acts as a pH indicator. As dermatophytes preferentially metabolize proteins, they produce alkaline metabolites that turn the medium from yellow to red.

175. (D) Artifactual ice crystal formation disrupting cells
Freezing a tissue biopsy before placing it in formalin causes the water inside the cells to crystallize. This ice crystal formation creates severe freeze artifacts, characterized by microscopic vacuoles, cellular shrinkage, and physical disruption of the tissue architecture. These artifacts severely compromise the pathologist's ability to evaluate cellular details and make a diagnosis.

176. (C) Refraction of light through a liquid proportional to dissolved solute concentration
A refractometer operates on the physical principle of light refraction. When light passes from air into a liquid, it bends at an angle determined by the concentration of dissolved solutes in that fluid. Measuring this angle of refraction provides an estimate of total dissolved solids, allowing rapid determination of urine specific gravity.

177. (B) Spherocytosis, autoagglutination, and a positive Coombs test
Immune-mediated hemolytic anemia is characterized by the body's immune system targeting its own red blood cells for destruction. The combination of prominent spherocytosis, macroscopic or microscopic autoagglutination, and a positive direct Coombs test is highly pathognomonic, indicating antibody-mediated targeting, membrane remodeling by macrophages, and active erythrocyte clumping.

178. (D) Increased membrane lipid ratio from liver disease
Target cells, or codocytes, are erythrocytes with an excess of cell membrane relative to their hemoglobin content, causing a central area of hemoglobin condensation. This abnormal morphology is commonly observed in animals with liver disease due to an increased membrane cholesterol-to-phospholipid ratio, which alters the physical structure of the cell membrane.

179. (A) Parasitic migrations and hypersensitivity reactions
Eosinophilia is a classic hematological finding associated with active parasitic migrations, particularly helminths, and immediate hypersensitivity or systemic allergic reactions. Mast cells and T-lymphocytes release specific cytokines, such as interleukin-five, which directly stimulate the bone marrow to increase the production, maturation, and release of eosinophils into the systemic circulation.

180. (B) II only
Thrombocytopenia primarily manifests as signs of primary hemostatic failure, which include petechiae, ecchymoses, and bleeding from mucosal surfaces, such as epistaxis or hematuria. In contrast, large, deep tissue hematomas are characteristic of secondary hemostatic disorders, such as coagulation factor deficiencies or rodenticide toxicity, rather than platelet deficits.

181. (A) Granulocytic precursor to nucleated erythroid precursor ratio
The myeloid-to-erythroid ratio in bone marrow aspirates compares the relative numbers of granulocytic precursors to nucleated erythroid precursors. This ratio is a critical parameter for evaluating bone marrow activity, helping clinicians determine whether an abnormal peripheral blood count is due to increased, decreased, or ineffective production of specific cell lines.

182. (D) Disseminated Intravascular Coagulation
Disseminated intravascular coagulation is a complex, life-threatening syndrome characterized

Downloaded from www.pdf.tube

142. (C) Round cell tumor
Round cell tumors exfoliate highly cellular samples containing individual, discrete, round-to-oval cells that lack cohesive intercellular junctions. On a cytologic smear, these cells are arranged singly and display distinct, well-defined cytoplasmic margins. Common examples of this tumor class include mast cell tumors, lymphoma, plasmacytoma, histiocytoma, and transmissible venereal tumors.

143. (D) Squamous epithelial cell
Squamous epithelial cells represent the largest cellular elements routinely observed during microscopic evaluation of canine urine sediment. These cells originate from the distal urethra, vagina, or prepuce. Morphologically, they are characterized by their vast, thin, flat cytoplasm with sharp, angular, or folded edges, and a small, round, centrally located, pyknotic nucleus.

144. (C) Isosthenuria
Isosthenuria occurs when the urine specific gravity matches that of glomerular filtrate, typically between one point zero zero eight and one point zero one two. If an animal remains isosthenuric despite suffering from severe clinical dehydration, it indicates a loss of the kidney's physiological ability to concentrate or dilute urine.

145. (B) Transitional cell carcinoma (TCC)
Finding transitional epithelial cells with marked anaplasia, severe pleomorphism, increased mitotic figures, and multinucleation in a urine sample is highly indicative of transitional cell carcinoma. While normal cells can slough during catheterization, the presence of distinct malignant criteria within these cells points to a neoplastic process within the urinary tract.

146. (D) Diabetes insipidus
Diabetes insipidus is characterized by persistent hyposthenuria and severe polydipsia. A complete failure to concentrate urine during a water deprivation test confirms a lack of functional antidiuretic hormone action. This can stem from a deficiency in hormone production by the pituitary gland or a cellular resistance to the hormone within the kidneys.

147. (A) Fecal sedimentation
Trematode eggs are exceptionally large and heavy, making them difficult to recover using standard flotation techniques. Fecal sedimentation allows these heavy ova to sink to the bottom of a water or saline mixture, concentrating them in the sediment for microscopic examination, which is essential for diagnosing liver and intestinal flukes.

148. (C) Long, unjointed pedicels with terminal suckers
Sarcoptes scabiei is a burrowing mite that causes intense pruritic mange in dogs. Microscopic identification relies on its round, globular body, short legs, and the presence of long, unjointed pedicels with terminal suckers on the front pairs of legs. This feature distinguishes it from other mites like Psoroptes, which have jointed pedicels.

NODIA

149. (A) It allows evaluation of the transition zone from normal tissue to the edge of the ulcer.
A wedge biopsy is highly valuable for diagnosing ulcerative skin lesions because it captures the critical transition zone where diseased tissue meets healthy skin. This allows pathologists to evaluate changes along the active margin of the ulcer, providing essential clues regarding infectious, immune-mediated, or neoplastic etiologies that might be lost in necrotic areas.

150. (A) Both A and R are true and R is the correct explanation of A.
A total leukocyte count alone does not provide a complete assessment of an inflammatory response. A normal total count can easily mask severe, life-threatening abnormalities in specific cell lines, such as a profound neutropenia combined with a massive regenerative left shift, which is only revealed through a manual differential leukocyte count.

151. (B) Fixation in 10% neutral buffered formalin without over-fixation
Prompt fixation of tissue specimens in ten percent neutral buffered formalin is crucial for preserving antigenicity and tissue architecture for subsequent immunohistochemistry. While formalin stabilizes proteins through cross-linking, excessive prolonged exposure can lead to over-fixation and antigen masking, which may require specialized antigen retrieval steps prior to staining.

152. (C) Primary hemostasis
The buccal mucosal bleeding time is an in vivo diagnostic test used to evaluate primary

hemostasis. It measures the time required for a vascular plug to form following a standardized incision, reflecting platelet number, platelet function, and vascular integrity, while remaining independent of the coagulation cascade that drives secondary hemostasis.

153. (D) Iron deficiency anemia
Iron deficiency anemia classically presents with low serum iron concentrations coupled with an elevated total iron-binding capacity. This elevation occurs as the liver increases transferrin production in an attempt to maximize the transport of scarce iron resources. This pattern helps differentiate true iron deficiency from the anemia of chronic disease.

154. (A) Mechanical shearing by intravascular fibrin strands
Schistocytes are fragmented red blood cells that are formed when intact erythrocytes are physically sliced by fibrin strands suspended within microvessels. This mechanical shearing is a hallmark of microangiopathic processes, such as disseminated intravascular coagulation, hemangiosarcoma, or glomerulonephritis, where abnormal fibrin deposition occurs within the vascular lumen.

155. (B) Falsely elevated PCV due to trapped plasma
Insufficient centrifugation of a microhematocrit tube fails to completely pack the red blood cells at the bottom of the column. This leaves a significant volume of plasma trapped between the erythrocytes, leading to a falsely elevated packed cell volume reading that does not accurately reflect the animal's true red blood cell mass.

156. (B) Fractional percentage of celltype × Total Leukocyte Count
To determine the absolute value of a specific leukocyte type, the fractional percentage of that cell, obtained from a manual differential count, is multiplied by the total leukocyte count. Utilizing absolute values rather than percentages is essential for clinical interpretation, as relative percentages can be highly misleading in a fluctuating leukogram.

157. (A) Extreme non-neoplastic leukocytosis mimicking leukemia
A leukemoid response is characterized by an extremely high leukocyte count that mimics leukemia but is actually a benign, reactive response to severe inflammation. This hematological phenomenon is marked by a profound neutrophilia accompanied by a significant left shift, commonly triggered by localized pyogenic infections, abscesses, or immune-mediated diseases.

158. (C) Hyposegmentation of granulocyte nuclei with mature chromatin
Pelger-Huët anomaly is a benign, inherited genetic condition characterized by a failure of granulocytic nuclei to undergo normal segmentation. Microscopically, the neutrophils appear as band cells, metamyelocytes, or myelocytes, but they possess mature, highly condensed chromatin and function normally, which helps differentiate this anomaly from a true inflammatory left shift.

159. (D) Saline dispersion test
The saline dispersion test is utilized to differentiate true antibody-mediated erythrocyte autoagglutination from simple rouleaux formation. Mixing a drop of blood with several drops of physiological saline disperses the weak electrostatic bonds holding rouleaux together, whereas the strong covalent antibody bonds of true autoagglutination will remain clumped under microscopic evaluation.

160. (A) P-4, Q-3, R-1, S-2
Serum cobalamin is absorbed exclusively in the ileum, while folate absorption occurs in the proximal small intestine. Trypsin-like immunoreactivity is a highly sensitive and specific biomarker for diagnosing exocrine pancreatic insufficiency, whereas pancreatic lipase immunoreactivity serves as the gold-standard test for confirming acute or chronic pancreatitis in domestic patients.

161. (B) Factor VIII
Activated partial thromboplastin time measures the integrity of the intrinsic and common coagulation pathways, which include factors twelve, eleven, nine, eight, ten, five, two, and one. A prolonged partial thromboplastin time paired with a normal prothrombin time points to a selective deficiency in the intrinsic pathway, such as factor eight.

162. (D) Creatine Kinase (CK)
Creatine kinase is the most sensitive and specific serum enzyme biomarker for detecting skeletal

Click Here to Buy book on Amazon

muscle injury in domestic animals. This enzyme is highly concentrated within the cytoplasm of myofibers and is rapidly released into the systemic circulation following cell membrane damage, showing significant elevations even after minor muscular trauma.

163. (A) Kidney
Serum amylase is produced primarily by the exocrine pancreas and is subsequently cleared from circulation by the kidneys through glomerular filtration and tubular degradation. Consequently, decreased renal function or acute kidney injury reduces the clearance rate of this enzyme, leading to significant elevations in serum amylase levels without active pancreatic disease.

164. (C) Loss of oncotic pressure causing edema or effusions
Albumin is the primary protein responsible for maintaining intravascular colloid oncotic pressure. When serum albumin levels drop below one and a half grams per deciliter, the oncotic pressure becomes insufficient to hold fluid within the vascular space, leading to transudation of fluid into the interstitial tissues or body cavities, causing edema.

165. (A) K3-EDTA tube
Drawing blood into a potassium EDTA tube prior to biochemical analysis causes severe, artificial electrolyte imbalances. The ethylenediaminetetraacetic acid chelates calcium ions, leading to profoundly low calcium readings, while the potassium salt additive in the anticoagulant introduces high levels of potassium, resulting in a false diagnosis of extreme hyperkalemia.

166. (D) Cytocentrifugation
Cytocentrifugation is a specialized preparation technique designed specifically for processing fluids with very low cellular concentrations, such as cerebrospinal fluid. This method utilizes centrifugal force to gently deposit cells onto a small area of a glass slide while simultaneously absorbing the fluid matrix, yielding highly concentrated, monolayer smears for accurate evaluation.

167. (B) Neutrophils and Macrophages
A pyogranulomatous inflammatory response is cytologically characterized by the co-presence of significant numbers of both neutrophils and activated macrophages, with macrophages often representing more than fifteen percent of the total inflammatory cell population. This specific cellular mixture is typically associated with chronic infections, foreign body reactions, or fungal diseases.

168. (C) Marked anisokaryosis, multiple nucleoli, and mitotic figures
Identifying a malignant mesenchymal tumor requires observing multiple cellular criteria of malignancy within the spindle cell population. Significant indicators of malignancy include marked anisokaryosis, the presence of multiple or prominent nucleoli, high mitotic activity with abnormal mitotic figures, and a high nuclear-to-cytoplasmic ratio, which are absent in benign fibromas.

169. (D) Precipitation of Tamm-Horsfall mucoprotein in acid urine
Hyaline casts are formed when Tamm-Horsfall mucoprotein precipitates within the lumen of the distal renal tubules or collecting ducts. This process is favored by acidic urine conditions, high solute concentrations, and a slow tubular flow rate, resulting in cylindrical, semi-transparent structures that are subsequently flushed out and observed in urine sediment.

170. (A) I and II
Struvite crystals frequently form in alkaline urine and are often associated with urinary tract infections caused by urease-producing bacteria in dogs. Additionally, uric acid crystals are considered a normal finding in the urine of healthy Dalmatians due to their unique, inherited hepatic and renal defect in uric acid metabolism.

NODIA

171. (B) Trichomonas spp.
Microscopic observation of motile, pear-shaped protozoa equipped with flagella and a characteristic undulating membrane in a fresh urine sample indicates infection with Trichomonas species. These flagellates can contaminate the urinary tract from the genital or gastrointestinal tracts, and their rapid, jerky movement is highly diagnostic on wet mount preparations.

172. (B) Steatorrhea
Steatorrhea is the specific clinical term utilized to describe the presence of excess, undigested fat within the feces. This condition typically arises from maldigestion, such as exocrine pancreatic

osmotic lysis or distortion that occurs when using standard formalin or alcohol solutions.

193. (C) 1.250 to 1.300
Standard veterinary sucrose flotation solution is formulated to a specific gravity of approximately one point two five zero to one point three zero zero. This high specific gravity is essential to exceed the density of common nematode and cestode ova, allowing them to float to the surface for collection.

194. (D) Feline Hemotropic Mycoplasmosis
Feline hemotropic mycoplasmosis is a parasitic disease caused by hemotropic mycoplasmas, such as Mycoplasma haemofelis, which attach to the surface of feline erythrocytes. This epicellular colonization triggers immune-mediated destruction of the red blood cells by splenic macrophages, leading to severe, life-threatening regenerative hemolytic anemia in affected cats.

195. (A) Small, round to signet-ring-shaped piroplasms
Cytauxzoon felis is a highly pathogenic protozoal parasite of cats. During the erythrocytic stage of infection, the organism is observed microscopically within red blood cells as small, round, oval, or signet-ring-shaped piroplasms. These distinct intracellular structures are typically one to two micrometers in diameter, helping confirm a diagnosis.

196. (C) Immunoglobulin free light chains
Bence Jones proteins are monoclonal immunoglobulin free light chains produced in excessive quantities by neoplastic plasma cells in patients with multiple myeloma. Due to their small molecular weight, these light chains are easily filtered by the glomerulus and enter the urine, where they serve as a critical diagnostic marker for myeloma.

197. (A) The reagent pad detects albumin and has very poor sensitivity for light chains.
Standard urine reagent dipsticks utilize a colorimetric protein error of indicators method that is highly sensitive to albumin but has extremely low sensitivity for globulins and immunoglobulin light chains. Consequently, patients with multiple myeloma secreting high concentrations of Bence Jones proteins often yield falsely negative protein results on routine dipstick tests.

198. (D) Mean Corpuscular Hemoglobin (MCH)
Mean corpuscular hemoglobin measures the average absolute weight of hemoglobin contained within a single erythrocyte. Calculated by dividing the total hemoglobin concentration by the red blood cell count, this index is expressed in picograms and provides valuable information regarding hemoglobin synthesis, complementing mean corpuscular volume and concentration.

199. (B) Bone marrow injury, splenic disease, or lead toxicity
The presence of nucleated red blood cells in peripheral blood without concurrent reticulocytosis is an inappropriate response. This indicates a breakdown in the bone marrow barrier, which can stem from direct bone marrow injury, splenic disease failing to clear/extract immature cells, or lead toxicity, which inhibits enzymes required for hemoglobin synthesis.

200. (A) Both A and R are true and R is the correct explanation of A.
Elevated serum amylase and lipase are not pathognomonic for acute pancreatitis. These enzymes are also synthesized by extrapancreatic tissues, including the stomach and duodenum, and are cleared from the circulation by the kidneys. Therefore, decreased renal clearance due to kidney disease can cause non-specific, artifactual elevations of both enzymes.

201. (D) Alkaline Phosphatase (ALP)
Alkaline phosphatase exists as several tissue-specific isoenzymes in dogs. Exposure to high levels of endogenous or exogenous glucocorticoids induces the synthesis of a unique, heat-stable, steroid-induced alkaline phosphatase isoenzyme. This induction can cause dramatic increases in total serum alkaline phosphatase activity, serving as a useful diagnostic indicator.

202. (A) Serum Ferritin
Serum ferritin is an iron-phosphorus-protein complex that serves as the primary intracellular iron-storage molecule. Because circulating ferritin levels correlate directly with total body iron stores, measuring serum ferritin is the most sensitive and reliable non-invasive diagnostic method for detecting early depletion of iron reserves before clinical iron deficiency anemia develops.

Downloaded from www.pdf.tube

by systemic activation of coagulation, leading to microvascular thrombosis and consumption of clotting factors and platelets. This is confirmed by the combination of prolonged PT and aPTT, severe thrombocytopenia, and elevated fibrin degradation products, indicating active, extensive fibrinolysis.

183. (B) Hyponatremia and Hyperkalemia
Hypoadrenocorticism is characterized by a deficiency of aldosterone, a hormone responsible for promoting renal sodium reabsorption and potassium excretion. A lack of aldosterone leads to excessive urinary sodium loss and potassium retention, resulting in the classic electrolyte pattern of hyponatremia and hyperkalemia, which often displays a low sodium-to-potassium ratio.

184. (C) Metabolic acidosis
Measuring the anion gap is a clinical tool used to classify the etiology of metabolic acidosis. A high anion gap indicates an accumulation of unmeasured organic anions, such as lactate, ketones, or uremic toxins, whereas a normal anion gap points to a primary loss of bicarbonate ions, such as with diarrhea.

185. (D) Triglyceride concentration in the fluid significantly higher than in the serum
Confirming a chylous pleural or peritoneal effusion requires demonstrating that the triglyceride concentration in the collected fluid is significantly higher than that in concurrent serum. Chyle is rich in chylomicrons containing triglycerides absorbed from the intestinal tract, which typically makes the fluid appear milky and elevated in lipid content.

186. (A) Sudan IV
Sudan IV is a fat-soluble dye that binds specifically to neutral lipids and triglycerides. When applied to cytological preparations, such as a liver fine needle aspirate, it stains intracellular lipid droplets a brilliant orange-red color. This staining technique is uniquely valuable for confirming hepatic lipidosis and differentiating fat from glycogen.

187. (B) Ketonuria
A strong, distinct sweet or fruity odor in canine urine is a classic clinical sign of ketonuria. Ketone bodies, particularly acetoacetate and acetone, are volatile compounds excreted in the urine during periods of excessive fat metabolism, which commonly occurs in patients suffering from diabetic ketoacidosis or prolonged starvation.

188. (B) Rapid lateral flow immunochromatographic strip tests
Rapid lateral flow immunochromatographic strip tests utilize capillary action to carry a liquid sample across a membrane embedded with specific antibodies. When target antigens are present, they bind to labeled antibodies, forming visible colored lines. This technology provides rapid, point-of-care detection for various infectious pathogens in veterinary clinical medicine.

189. (A) Normal distal urethra or genital tract contamination
Squamous epithelial cells line the distal urethra, vagina, and prepuce. Finding numerous squamous cells in a free-catch urine sample is a common and normal finding, representing benign contamination from these lower urogenital areas as the urine is voided. It does not indicate upper urinary tract disease or renal damage.

190. (A) I only
Normal cerebrospinal fluid is ultrafiltered plasma containing minimal fibrinogen and protein, meaning it should never clot upon standing. The formation of a clot in a cerebrospinal fluid sample indicates a significant breakdown of the blood-brain barrier, which allows fibrinogen to leak into the fluid, or iatrogenic contamination with blood.

NODIA

191. (D) Replace dehydrating alcohol with a wax-miscible solvent
During histopathological tissue processing, clearing is the step that replaces the dehydrating agent, typically alcohol, with a solvent that is miscible with embedding media, such as paraffin wax. Common clearing agents include xylene or toluene, which render the tissue transparent and prepare it for complete paraffin infiltration and block casting.

192. (C) Polyvinyl Alcohol or Sodium Acetate-Acetic Acid-Formalin
Preserving fecal samples for subsequent protozoan cyst evaluation requires specialized fixatives like polyvinyl alcohol or sodium acetate-acetic acid-formalin. These solutions stabilize the delicate internal structures and external membranes of fragile trophozoites and cysts, preventing

CHAPTER 09
Systemic Pathology

2022

1. Which of the following congenital heart defects is characterized by a left-to-right shunt that often leads to pulmonary hypertension, right ventricular hypertrophy, and a subsequent reversal of the shunt (Eisenmenger syndrome)?
(A) Pulmonic stenosis
(B) Ventricular septal defect
(C) Tetralogy of Fallot
(D) Persistent right aortic arch

2008

2. In the pathogenesis of traumatic reticulopericarditis in bovines, the morphological classification of the typical pericardial lesion is best described as which of the following?
(A) Serous pericarditis
(B) Purulent pericarditis
(C) Granulomatous pericarditis
(D) Fibrinous pericarditis

2022

3. In Virchow's triad, which of the following is the most significant primary determinant of thrombosis in cases of strongyle-induced cranial mesenteric arteritis in horses?
(A) Endothelial injury
(B) Systemic hypercoagulability
(C) Stasis of blood flow
(D) Increased blood viscosity

2005

4. Splenic infarcts characteristically present as wedge-shaped, well-demarcated lesions at the organ periphery. The classical presentation of multiple marginal splenic infarcts is highly indicative of which infectious disease in swine?
(A) African Swine Fever
(B) Porcine Reproductive and Respiratory Syndrome
(C) Classical Swine Fever
(D) Porcine Circovirus type 2

2011

5. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).
Assertion (A): Thymomas are frequently associated with secondary immune-mediated conditions such as myasthenia gravis and exfoliative dermatitis.
Reason (R): Thymomas arise from the lymphoid cellular component of the thymus, leading to direct production of autoantibodies.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2012

6. Tracheal collapse in toy breed dogs typically involves dorsoventral flattening of the trachea. Which structural alteration is primarily responsible for this pathology?
(A) Chronic hyperplastic mucosal changes of the pseudostratified epithelium
(B) Extensive mineralization and ossification of the dorsal tracheal membrane
(C) Diffuse infiltration of the submucosa by eosinophils and mast cells
(D) Loss of glycosaminoglycans and hypocellularity in tracheal rings

2011

7. Cranioventral consolidation of the lungs with distinct lobular patterns, fibrin deposition, and oat cell infiltration is the hallmark of which morphological classification of pneumonia?
(A) Interstitial pneumonia
(B) Fibrinous bronchopneumonia
(C) Granulomatous pneumonia
(D) Embolic pneumonia

2010

8. In the pathogenesis of Acute Respiratory Distress Syndrome (ARDS), the fundamental structural lesion that impairs gas exchange is identified as which of the following?
(A) Severe hyperplastic response of the bronchiolar epithelium
(B) Accumulation of suppurative exudate within alveolar spaces
(C) Diffuse alveolar damage with hyaline

Click Here to Buy book on Flipkart

203. (B) Septic exudate
A septic exudate is characterized by a high nucleated cell count exceeding five thousand cells per microliter, a high total protein concentration over three grams per deciliter, and a cytological predominance of degenerate neutrophils. Finding intracellular bacteria within these neutrophils confirms the presence of active, localized infectious sepsis within the cavity fluid.

204. (B) Impression smear
An impression smear, or imprint, is prepared by pressing a clean glass slide directly against the freshly cut surface of a tissue specimen or lesion. Prior to making the imprint, the tissue surface must be blotted dry to remove excess blood, allowing cells to adhere directly to the glass and preserving cellular architecture.

205. (A) P-2, Q-1, R-4, S-3
Potassium EDTA is the standard anticoagulant for mammalian complete blood counts, preserving cellular morphology. Sodium citrate is used for coagulation profiles because it preserves clotting factors. Lithium heparin is ideal for biochemistry and avian hematology, while sodium fluoride serves to inhibit glycolysis, preserving blood glucose levels for long-term storage.

206. (C) Davidson's fixative or Bouin's solution
Submitting a whole eye for histopathology requires specialized fixatives like Davidson's fixative or Bouin's solution. Standard ten percent formalin penetrates ocular tissues too slowly, causing retinal detachment and cellular degradation. Davidson's fixative penetrates rapidly, minimizing artifacts and preserving the delicate relationships between the retina, choroid, and sclera.

207. (D) Myoglobinuria from rhabdomyolysis
Myoglobinuria occurs when severe skeletal muscle damage, or rhabdomyolysis, releases large amounts of myoglobin into the circulation. This small, iron-containing protein is rapidly filtered by the glomerulus, causing dark brown urine. This urine tests positive for occult blood on a dipstick but lacks intact erythrocytes under microscopic evaluation.

208. (A) Both A and R are true and R is the correct explanation of A.
Diagnosing motile protozoal trophozoites, such as Tritrichomonas foetus, requires immediate evaluation of a fresh direct fecal smear. The high osmotic pressure and specific gravity of standard flotation solutions cause rapid dehydration and osmotic lysis of these delicate, non-encysted life stages, rendering them completely unrecognizable under microscopic examination.

209. (B) Flaccid paralysis
Parturient paresis, or milk fever, is a disease of lactating dairy cows characterized by profound hypocalcemia. In cattle, low extracellular calcium levels block the release of acetylcholine at the neuromuscular junction, leading to a loss of muscle tone and subsequent flaccid paralysis, rather than the tetany observed in other species.

210. (A) P-2, Q-3, R-4, S-1
Heinz bodies represent precipitated, denatured hemoglobin caused by oxidative damage. Howell-Jolly bodies are nuclear remnants in red cells. Basophilic stippling consists of aggregated ribosomes indicating active erythroid regeneration or lead toxicity, while Döhle bodies are aggregates of rough endoplasmic reticulum and ribosomal RNA observed inside toxic neutrophils.

****

Downloaded from www.pdf.tube

132. (C) Large size, vacuolated blue-gray cytoplasm, and lobulated nucleus
Monocytes are the largest leukocytes found in peripheral blood smears. They are cytologically distinguished by their abundant, diffuse, blue-gray cytoplasm, which often contains small vacuoles, combined with a highly variable, pleomorphic, or lobulated nucleus. This irregular nuclear shape and abundant vacuolated cytoplasm help differentiate them from mature neutrophils and lymphocytes.

133. (A) Hypersegmented neutrophils
A right shift in a leukogram is defined as an increase in the number of hypersegmented neutrophils circulating in the blood. These cells represent aged neutrophils that have remained in circulation longer than normal, typically due to corticosteroid administration, which downregulates adhesion molecules and delays normal tissue emigration.

134. (D) Accelerated thrombopoiesis, as new platelets are larger
Mean platelet volume measures the average size of circulating platelets. An elevated volume indicates the presence of larger, juvenile platelets, which are released prematurely by the bone marrow during periods of active regeneration. This index is crucial for distinguishing between regenerative thrombocytopenia and bone marrow hypoplasia.

135. (B) Surra
Trypanosoma evansi is a pathogenic flagellated protozoan that causes Surra, a major vector-borne disease affecting horses, camels, dogs, and livestock. Transmitted mechanically by biting flies, the disease leads to severe systemic signs including progressive weakness, recurrent fever, anemia, edema, and significant mortality if left untreated.

136. (C) Extracellularly in the plasma
Trypanosoma evansi is a flagellated, spindle-shaped protozoal parasite that resides and multiplies extracellularly within the host's bloodstream. When examining a peripheral blood smear from an infected animal, these motile organisms are observed swimming freely in the plasma between erythrocytes, rather than invading the intracellular space of host cells.

137. (D) High AST and High CK
Creatine kinase is a highly sensitive and specific biomarker for skeletal muscle damage, rising rapidly after myofiber injury. Aspartate aminotransferase is also present in high concentrations within skeletal muscle and rises alongside creatine kinase during muscle necrosis, providing a classic biochemical pattern indicative of severe rhabdomyolysis.

138. (A) Unconjugated bilirubin bound to albumin
Prior to hepatic uptake, the majority of circulating bilirubin is in its unconjugated, lipid-soluble form. Because unconjugated bilirubin is highly hydrophobic, it must bind tightly to plasma albumin to be safely transported through the systemic circulation to the liver, where it is subsequently conjugated with glucuronic acid.

139. (B) An incorrectly calibrated refractometer prism
An incorrectly calibrated refractometer directly compromises the accuracy of total protein measurements, leading to artifactual hypoproteinemia or hyperproteinemia. While lipemia and hyperglycemia can cause falsely elevated total protein readings by altering light refraction, a physical calibration error on the prism prevents the instrument from establishing a correct zero baseline.

140. (A) Isolate mass → Insert needle with negative pressure → Release pressure → Withdraw needle → Expel onto slide
Performing a fine needle aspiration biopsy requires isolating the target mass, inserting the needle, and applying negative syringe pressure to harvest cells. Crucially, the negative pressure must be fully released before withdrawing the needle to prevent pulling the cellular sample into the syringe barrel, allowing successful expulsion onto a slide.

NODIA

141. (A) Cohesive clusters or sheets with distinct cell borders
Epithelial cells naturally possess strong intercellular junctions, which are preserved during cytological preparation. On a microscopic smear, these cells typically present as tightly cohesive clusters, sheets, or cords with clearly defined, distinct cell-to-cell borders, differentiating them from the individually exfoliated cells characteristic of mesenchymal or round cell tumors.

membrane formation
(D) Chronic granulomatous infiltration of the alveolar septa

2019
9. In the development of ruminal acidosis, the primary shift in ruminal microflora leads to an excessive production of which acid, causing the characteristic chemical ruminitis?
(A) Acetic acid
(B) Propionic acid
(C) Butyric acid
(D) Lactic acid

2017
10. The classic macroscopic lesion of Johne's disease in cattle is a thickened, corrugated intestinal mucosa. This structural alteration is driven primarily by the massive infiltration of which inflammatory cell type?
(A) Macrophages
(B) Neutrophils
(C) Eosinophils
(D) Lymphocytes

2001
11. Centrilobular hepatic necrosis is most frequently associated with which pathophysiological mechanism?
(A) Direct action of toxins absorbed from the gastrointestinal tract via the portal vein
(B) Severe hypoxia or passive venous congestion due to right-sided heart failure
(C) Biliary obstruction leading to bile salt toxicity
(D) Ascending bacterial infection from the duodenum

2005
12. In acute hemorrhagic necrotic pancreatitis, the severe local tissue destruction is primarily initiated by the premature intrapancreatic activation of which zymogen?
(A) Chymotrypsinogen
(B) Trypsinogen
(C) Proelastase
(D) Prolipase

2001
13. When comparing ischemic acute tubular necrosis (ATN) to toxic ATN, which of the following features is most characteristic of ischemic injury?
(A) Tubulorrhexis and multifocal tubular damage
(B) Intact tubular basement membranes
(C) Lesions strictly confined to the proximal convoluted tubule
(D) Rapid and complete regeneration of tubular epithelium without fibrosis

2014
14. Pulpy kidney disease in sheep is characterized by bilateral symmetrical encephalomalacia and rapid autolysis of the renal cortex. The pathogenesis is primarily mediated by which specific toxin?
(A) Alpha toxin of Clostridium perfringens type A
(B) Beta toxin of Clostridium perfringens type C
(C) Epsilon toxin of Clostridium perfringens type D
(D) Tetanospasmin of Clostridium tetani

2008
15. Match the specific urinary tract pathology in List I with its primary causative factor or mechanism in List II.
List I: (Pathology)
P. Feline Lower Urinary Tract Disease (FLUTD)
Q. Emphysematous cystitis
R. Enzootic hematuria
S. Struvite urolithiasis in dogs
List II: (Mechanism)
1. Chronic ingestion of bracken fern
2. Urease-producing bacterial infections
3. Idiopathic mechanisms involving matrix plugs
4. Glucosuria combined with gas-producing bacteria
(A) P-3, Q-4, R-1, S-2
(B) P-2, Q-1, R-4, S-3
(C) P-3, Q-2, R-4, S-1
(D) P-1, Q-4, R-2, S-3

2016
16. In the canine pyometra complex, the initial pathological change predisposing the uterus to secondary bacterial infection is which of the following?
(A) Estrogen-induced endometrial atrophy
(B) Prostaglandin-induced myometrial hypercontractility
(C) Progesterone-mediated cystic endometrial hyperplasia
(D) Oxytocin-mediated cervical dilation

2011
17. Benign prostatic hyperplasia (BPH) in older, intact male dogs is characterized macroscopically by symmetrical enlargement of the gland.

Click Here to Buy book on Amazon

2023

90. Which of the following statements identifies a key macroscopic difference between normal skeletal muscle and skeletal muscle undergoing nutritional myopathy (White Muscle Disease)?
(A) Affected muscle shows discrete pale, chalky-white streaks or patches.
(B) Affected muscle exhibits a diffuse yellow discoloration with fatty replacement.
(C) Affected muscle is uniformly dark red to black due to extensive hemorrhage.
(D) Affected muscle contains large, fluid-filled cysts.

2008

91. Primary hyperparathyroidism is a rare endocrine disorder. Which of the following is the most common pathological cause of primary hyperparathyroidism in dogs?
(A) Diffuse hyperplasia of all four parathyroid glands
(B) Functional adenoma of a single parathyroid gland
(C) Autoimmune destruction of parathyroid chief cells
(D) Metastatic carcinoma from the thyroid gland

2005

92. Hyperadrenocorticism (Cushing's disease) leads to widespread systemic effects. Which of the following dermatological lesions is classically associated with this endocrine pathology in dogs?
(A) Intense pruritus and hyperkeratosis
(B) Symmetrical bilateral alopecia and epidermal atrophy
(C) Papillary hypertrophy of the dermis
(D) Massive infiltration of the dermis by eosinophils

2012

93. In veterinary pathology, pheochromocytoma is a functional neoplasm originating from which specific anatomical structure?
(A) Adrenal medulla
(B) Adrenal cortex, zona fasciculata
(C) Pituitary pars distalis
(D) Pancreatic islets

2003

94. Corneal ulcers are a common ophthalmic pathology. A specific type of deep, rapidly progressing ulcer characterized by enzymatic destruction of the corneal stroma by proteases is known as which of the following?
(A) Indolent ulcer
(B) Descemetocele
(C) Melting ulcer (keratomalacia)
(D) Superficial punctate keratitis

2021

95. Match the specific epidermal or dermal response in List I with its accurate definition in List II.
List I: (Response)
P. Acanthosis
Q. Hyperkeratosis
R. Parakeratosis
S. Exocytosis
List II: (Definition)
1. Retention of nuclei in the stratum corneum
2. Migration of inflammatory cells into the epidermis
3. Increased thickness of the stratum spinosum
4. Thickening of the stratum corneum
(A) P-3, Q-4, R-1, S-2
(B) P-4, Q-3, R-2, S-1
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-3, S-2

2017

96. Acantholysis is an important pathological mechanism in certain autoimmune skin diseases, such as pemphigus foliaceus. Which of the following defines acantholysis?
(A) Premature death of individual keratinocytes
(B) Excessive production of epidermal melanin
(C) Loss of cohesion between epidermal cells
(D) Fibrous thickening of dermal-epidermal junction

NODIA

2015

97. In the context of left-sided congestive heart failure, which of the following represents the most direct pathophysiological consequence affecting the respiratory system?
(A) Chronic hepatic passive congestion
(B) Pulmonary hypertension and edema
(C) Progressive abdominal ascites
(D) Prominent jugular vein distension

2014

98. Cor pulmonale is defined as right ventricular enlargement (hypertrophy and/or dilation) secondary to which of the following primary conditions?
(A) Chronic left-sided congestive heart failure
(B) Congenital stenotic lesion of the pulmonic valve
(C) Severe sustained systemic arterial hypertension
(D) Primary pulmonary or pulmonary vascular

2018

26. Which of the following compensatory mechanisms in chronic congestive heart failure leads directly to increased blood volume and subsequent edema?
(A) Activation of the renin-angiotensin-aldosterone system
(B) Downregulation of sympathetic nervous system activity
(C) Increased release and activity of atrial natriuretic peptide
(D) Marked reduction in systemic antidiuretic hormone release

2003

27. Vegetative endocarditis most commonly affects the heart valves. In cattle, which valve is statistically the most frequently affected by bacterial endocarditis?
(A) Mitral valve
(B) Tricuspid valve
(C) Aortic valve
(D) Pulmonary valve

2022

28. Atherosclerosis is relatively rare in domestic animals compared to humans, but it is well-documented in which of the following species secondary to hypothyroidism?
(A) Feline
(B) Canine
(C) Bovine
(D) Equine

2004

29. Lymphadenitis is characterized by inflammation of the lymph nodes. In caseous lymphadenitis of sheep and goats, the typical "onion-ring" appearance of the lesion is caused by which pathogen?
(A) Corynebacterium pseudotuberculosis
(B) Mycobacterium bovis
(C) Streptococcus equi
(D) Actinobacillus lignieresii

2015

30. Which of the following represents the correct sequence of cellular events in the pathogenesis of acute local inflammation leading to purulent exudate?
(A) Margination → Vasodilation → Chemotaxis → Diapedesis → Phagocytosis
(B) Vasodilation → Chemotaxis → Margination → Diapedesis → Phagocytosis
(C) Vasodilation → Margination → Diapedesis → Chemotaxis → Phagocytosis
(D) Diapedesis → Vasodilation → Margination → Phagocytosis → Chemotaxis

2005

31. Aplastic anemia involves pancytopenia due to bone marrow failure. Which of the following toxic agents is famously associated with inducing aplastic anemia in calves through the ingestion of its active principle?
(A) Sweet clover
(B) St. John's wort
(C) Lantana camara
(D) Bracken fern

2009

32. In cases of atrophic rhinitis in swine, the primary pathological lesion is the destruction and atrophy of the nasal turbinates. This process is synergistically driven by the toxins of Bordetella bronchiseptica and which other organism?
(A) Mycoplasma hyopneumoniae
(B) Actinobacillus pleuropneumoniae
(C) Pasteurella multocida
(D) Haemophilus parasuis

2009

33. Granulomatous pneumonia is classically observed in systemic mycotic infections. Which of the following fungi typically produces a granulomatous to pyogranulomatous response in the lungs of dogs, often accompanied by broad-based budding yeast structures?
(A) Cryptococcus neoformans
(B) Blastomyces dermatitidis
(C) Histoplasma capsulatum
(D) Aspergillus fumigatus

2014

34. Alveolar emphysema is characterized by abnormal permanent enlargement of the air spaces distal to the terminal bronchioles. In horses with recurrent airway obstruction (heaves), the emphysema is primarily which of the following morphological types?
(A) Centriacinar emphysema
(B) Interstitial emphysema
(C) Paraseptal emphysema
(D) Panacinar emphysema

2004

35. Consider the following statements regarding oral and forestomach pathologies:
I. Vesicular stomatitis in cattle is clinically indistinguishable from Foot and Mouth Disease.
II. Primary ruminal tympany (frothy bloat)

Downloaded from www.pdf.tube

(C) Central or nephrogenic diabetes insipidus
(D) Primary immune-mediated hypothyroidism

2021
54. Hypoadrenocorticism (Addison's disease) in dogs most commonly results from which underlying pathological process affecting the adrenal cortex?
(A) Bilateral adrenal cortical hyperplasia
(B) Chronic granulomatous adrenalitis
(C) Metastatic neoplastic infiltration
(D) Immune-mediated cortical atrophy

2003
55. Match the specific ophthalmic or otic pathology in List I with its defining morphological characteristic in List II.
List I: (Pathology)
P. Cataract
Q. Keratitis
R. Otitis media
S. Glaucoma
List II: (Characteristic)
1. Inflammation of the tympanic bulla
2. Optic disc cupping due to high intraocular pressure
3. Loss of transparency of the lens
4. Neovascularization and inflammation of the cornea

(A) P-3, Q-4, R-1, S-2
(B) P-4, Q-3, R-2, S-1
(C) P-3, Q-1, R-4, S-2
(D) P-2, Q-4, R-1, S-3

2016
56. Spongiosis is a diagnostic histopathological finding in certain dermatological conditions. Which of the following defines spongiosis?
(A) Intracellular vacuolization of keratinocytes
(B) Thickening of the stratum granulosum
(C) Intercellular edema within the epidermis
(D) Premature keratinization of individual cells

2012
57. Which of the following describes the most relevant gross pathological feature of a patent ductus arteriosus (PDA) in a neonate?
(A) A communicative opening between the right and left ventricles
(B) A stricture of the aortic arch distal to the subclavian artery
(C) An anomalous origin of the right subclavian vessel from the aorta
(D) A persistent shunt between pulmonary artery and descending aorta

2018
58. Myocarditis in domestic animals is frequently viral in origin. Parvovirus infection in neonatal puppies classically causes which specific type of myocarditis?
(A) Interstitial myocarditis with intranuclear inclusions
(B) Suppurative myocarditis with abscessation and necrosis
(C) Eosinophilic myocarditis with severe hypersensitivity
(D) Granulomatous myocarditis with giant cell infiltration

2018
59. Aneurysms are focal abnormal dilations of a blood vessel. In horses, verminous aneurysms are characteristically found in the cranial mesenteric artery due to the migration of which parasite?
(A) Parascaris equorum
(B) Strongylus vulgaris
(C) Strongylus edentatus
(D) Oxyuris equi

2010
60. Phlebitis refers to inflammation of a vein. Omphalophlebitis, a common pathology in neonatal farm animals, specifically involves inflammation of which structure?
(A) Jugular vein
(B) Umbilical vein
(C) Portal vein
(D) Caudal vena cava

2011
61. Splenomegaly with a "meaty" consistency on cut surface, rather than bloody, is most indicative of which underlying pathophysiological process?
(A) Diffuse granulomatous or lymphoid hyperplasia
(B) Acute congestion secondary to barbiturate use
(C) Passive venous congestion due to splenic torsion
(D) Acute septicemic splenomegaly from anthrax

NODIA

2005
62. In feline leukemia virus (FeLV) infection, the virus primarily induces malignant transformation leading to which of the following neoplastic conditions?
(A) Multiple myeloma
(B) Histiocytic sarcoma
(C) T-cell lymphoma
(D) Cutaneous plasmacytoma

(C) Staphylococcus aureus
(D) Mycoplasma bovis

2006

45. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).
Assertion (A): In Wallerian degeneration, the distal portion of a severed axon undergoes complete fragmentation and phagocytosis.
Reason (R): The distal axon is separated from the neuronal cell body, depriving it of essential proteins and organelles transported via axoplasmic flow.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2023

46. Neuronophagia is a specific microscopic finding indicative of severe neuronal damage. Which cell type is primarily responsible for engulfing the necrotic neuron in this process?
(A) Astrocytes
(B) Oligodendrocytes
(C) Microglia
(D) Ependymal cells

2001

47. Viral encephalitis often presents with distinct inclusion bodies. In rabies infection, the pathognomonic Negri bodies are intracytoplasmic inclusions found most prominently in which neurons of the canine brain?
(A) Purkinje cells of the cerebellum
(B) Motor neurons of the ventral horn
(C) Neurons of the substantia nigra
(D) Pyramidal cells of the hippocampus

2013

48. Rickets and osteomalacia are both characterized by a failure of bone mineralization. What is the fundamental difference in the pathogenesis of these two conditions?
(A) Rickets occurs only in carnivores, whereas osteomalacia is restricted to herbivores.
(B) Rickets is caused exclusively by excessive dietary calcium, while osteomalacia is caused by vitamin D toxicity.
(C) Rickets involves failure of mineralization of both the physis and osteoid in growing animals, whereas osteomalacia involves failure of osteoid mineralization in adults with closed physes.
(D) Rickets is an inflammatory disease of the joint capsule, whereas osteomalacia is a primary neoplasm of osteoblasts.

2015

49. Osteochondrosis is a focal failure of endochondral ossification. In swine, which specific joint is most frequently affected by osteochondrosis dissecans?
(A) Stifle (femorotibial) joint
(B) Elbow (humeroradial) joint
(C) Shoulder (scapulohumeral) joint
(D) Carpal joint

2025

50. Myositis ossificans is a pathological condition characterized by which of the following?
(A) Bacterial infection of skeletal muscle leading to extensive gas formation
(B) Immune-mediated destruction of muscle fibers
(C) Congenital absence of specific muscle groups
(D) Metaplastic bone formation within injured skeletal muscle

2005

51. In horses, tying-up syndrome (exertional rhabdomyolysis) is morphologically characterized by which of the following lesions?
(A) Zenker's degeneration and necrosis of myofibers
(B) Diffuse chronic granulomatous inflammation of muscle
(C) Acute suppurative inflammation with abscess formation
(D) Extensive interstitial fibrosis and fat infiltration

2014

52. Secondary hyperparathyroidism of nutritional origin is most classically seen in animals fed a diet characterized by which of the following mineral imbalances?
(A) High calcium, low phosphorus
(B) High calcium, high magnesium
(C) Low calcium, high phosphorus
(D) Low calcium, low potassium

2014

53. Pituitary adenomas originating from the pars intermedia are commonly diagnosed in older horses. This condition is clinically recognized as which of the following?
(A) Pituitary Pars Intermedia Dysfunction
(B) Equine peripheral metabolic syndrome

Click Here to Buy book on Flipkart

is usually related to the ingestion of large quantities of grain.
III. Secondary ruminal tympany (free-gas bloat) involves a physical or functional obstruction of eructation.
IV. Fusobacterium necrophorum is a primary causative agent of viral stomatitis.
Which of the above statements are incorrect?
(A) I and II
(B) II and IV
(C) I, III and IV
(D) I and III

2016
36. Left displaced abomasum (LDA) in dairy cattle is most strongly associated with which physiological period?
(A) Active mid-lactation
(B) Late gestation period
(C) Early postpartum weeks
(D) Extended dry period

2013
37. Gastric ulcers in swine are a common pathology. The lesions are overwhelmingly located in which specific anatomical region of the porcine stomach?
(A) Pars esophagea
(B) Fundic region
(C) Pyloric antrum
(D) Cardia region

2003
38. Regarding hepatic injury, midzonal hepatic necrosis is an uncommon pattern but is classically described in horses or pigs affected by which of the following?
(A) Chronic right heart failure
(B) Pyrrolizidine alkaloid toxicity
(C) Anemia or severe hypovolemic shock
(D) Aflatoxicosis

2015
39. Exocrine pancreatic insufficiency (EPI) in dogs is most commonly caused by which underlying pathological process?
(A) Chronic recurrent pancreatitis
(B) Pancreatic acinar atrophy
(C) Pancreatic duct obstruction by calculi
(D) Exocrine pancreatic adenocarcinoma

2022
40. In evaluating chronic renal failure mechanisms, the term "end-stage kidney" refers to a morphological state where the primary insult is no longer recognizable. Which triad of features definitively characterizes an end-stage kidney?
(A) Cellular hypertrophy, active hyperemia, and fluid exudation
(B) Interstitial edema, hemorrhage, and tubular hyperplasia
(C) Fibrosis, mineralization, and functional parenchymal loss
(D) Active suppuration, cyst formation, and systemic amyloidosis

2023
41. Glomerulonephritis is primarily an immune-mediated disease. Which fundamental mechanism is responsible for the majority of naturally occurring glomerulonephritis cases in domestic animals?
(A) Direct cytotoxic T-cell attack on podocytes
(B) Production of autoantibodies directed specifically against the glomerular basement membrane
(C) Ischemic damage due to afferent arteriole constriction
(D) Deposition of circulating immune complexes in the glomerular capillary walls

NODIA

2006
42. Urolithiasis is significantly influenced by urine pH. In dogs, which type of urolith is typically formed in alkaline urine, commonly secondary to a staphylococcal lower urinary tract infection?
(A) Struvite
(B) Calcium oxalate
(C) Urate
(D) Cystine

2018
43. The pathogenesis of abortion in bovine brucellosis is primarily driven by the affinity of Brucella abortus for the pregnant uterus. This tropism is largely due to the presence of which carbohydrate in the fetal fluids and placenta?
(A) Fructose
(B) Erythritol
(C) Galactose
(D) Sorbitol

2014
44. Among the classifications of mastitis, environmental mastitis is frequently acute and severe. Which of the following is the most common coliform pathogen responsible for acute gangrenous mastitis in dairy cattle?
(A) Streptococcus agalactiae
(B) Escherichia coli

2007

63. Sinusitis in horses frequently occurs as a secondary consequence of which primary pathology?
(A) Equine viral arteritis
(B) Guttural pouch mycosis
(C) Laryngeal hemiplegia
(D) Maxillary tooth root disease

2008

64. Morphological classification of pneumonias relies on the distribution of lesions. Interstitial pneumonia is characterized primarily by inflammation and thickening of which specific pulmonary structure?
(A) Bronchial mucosa
(B) Pleural surface
(C) Alveolar septa
(D) Terminal bronchioles

2002

65. Consider the following statements regarding pulmonary hemodynamic disorders:
I. Pulmonary edema is characterized by the accumulation of transudate in the alveoli.
II. Atelectasis refers to the abnormal distension of alveoli with air.
III. Heart failure cells in the lungs are hemosiderin-laden macrophages.
Which of the above statements are correct?
(A) I and III
(B) I and II
(C) II and III
(D) I only

2005

66. Pulmonary atelectasis can be classified based on its mechanism. Compression atelectasis is most commonly associated with which of the following conditions?
(A) Complete physical obstruction of a major bronchus
(B) Severe congenital loss of pulmonary surfactant
(C) Chronic fibrotic contraction of lung parenchyma
(D) Accumulation of fluid or air in the pleural cavity

2008

67. Ruminitis-liver abscess complex in cattle begins with ruminal acidosis. The typical entry of bacteria into the portal circulation occurs via which pathomechanism?
(A) Mucosal breach from chemical ruminitis
(B) Direct diaphragmatic penetration by foreign bodies
(C) Ascending duodenal infection into the bile duct
(D) Hematogenous spread from a primary pneumonia site

2013

68. The diagnostic lesion of abomasal displacement (LDA) on necropsy, aside from the anatomical malpositioning, often includes which secondary change?
(A) Transmural infarction and gangrene of the wall
(B) Diffuse granulomatous infiltration of the wall
(C) Mucosal congestion and superficial errors
(D) Rupture of the abomasum along the greater curvature

2010

69. Specific enteritis classifications include hemorrhagic, fibrinous, and necrotic. In swine, proliferative hemorrhagic enteropathy is caused by the intracellular bacterium Lawsonia intracellularis, which specifically targets which intestinal cells?
(A) Crypt epithelial cells
(B) Goblet cells
(C) Peyer's patch lymphocytes
(D) Brunner's gland acinar cells

2007

70. Consider the following events in the pathogenesis of hepatic cirrhosis:
I. Nodular regeneration of surviving hepatocytes
II. Chronic irreversible hepatocyte necrosis
III. Diffuse deposition of connective tissue (fibrosis)
IV. Portal hypertension
Which of the following represents the correct chronological order of these events?
(A) I, II, III, IV
(B) II, III, I, IV
(C) III, II, IV, I
(D) II, I, III, IV

2009

71. Periportal hepatic necrosis is classically caused by toxic substances that require no metabolism to be toxic. Which of the following best explains why the periportal zone is most severely affected?
(A) It possesses the lowest oxygen tension within the hepatic acinus.
(B) It receives the highest toxin concentration from incoming blood.

NODIA

This condition is fundamentally dependent on the conversion of testosterone to which active metabolite within the prostate?
(A) Androstenedione
(B) Dihydrotestosterone
(C) Estradiol
(D) Dehydroepiandrosterone

2016

18. Central chromatolysis is a distinct morphological response of neurons. Which of the following best describes the classical appearance of a neuron undergoing this process?
(A) Shrunken cell body with a pyknotic, centrally positioned nucleus
(B) Accumulation of lipofuscin pigments in the perinuclear cytoplasm
(C) Transformation of the neuronal body into a multinucleated giant cell
(D) Swollen cell body, peripheral nucleus, and dispersed Nissl substance

2025

19. Spongiform encephalopathies, such as BSE and Scrapie, present with distinct neuropathological features. Which of the following structural changes is absent in these diseases?
(A) Perivascular mononuclear cuffing
(B) Extensive vacuolation of neurons
(C) Severe reactive astrogliosis
(D) Deposition of abnormal prion protein

2009

20. Osteodystrophia fibrosa is a metabolic bone disease characterized by excessive bone resorption and replacement by fibrous connective tissue. This pathology is a direct consequence of which underlying endocrine state?
(A) Chronic primary hypothyroidism
(B) Idiopathic hypercalcitoninism
(C) Secondary hyperparathyroidism
(D) Primary hypoadrenocorticism

2022

21. In nutritional myopathy (White Muscle Disease), the underlying deficiency leads to oxidative damage of cellular membranes. Which specific organelle's membrane is primarily targeted, leading to calcium influx and subsequent myofiber necrosis?
(A) Sarcoplasmic reticulum
(B) Nucleus
(C) Golgi apparatus
(D) Rough endoplasmic reticulum

2001

22. In the pathogenesis of primary goiter due to iodine deficiency, the defining morphological response of the thyroid gland is best classified as which of the following?
(A) Diffuse follicular atrophy with stromal fibrosis
(B) Focal neoplastic transformation of thyroid C-cells
(C) Chronic granulomatous destruction of follicles
(D) Follicular cell hypertrophy and hyperplasia

2010

23. In feline diabetes mellitus, the morphological hallmark most frequently observed in the pancreatic islets of Langerhans is which of the following?
(A) Lymphocytic infiltration (insulitis)
(B) Islet amyloidosis
(C) Severe acute necrotizing pancreatitis
(D) Hyperplasia of the exocrine acini

2017

24. Glaucoma is characterized by a sustained increase in intraocular pressure. The primary pathological consequence leading to irreversible blindness in chronic glaucoma is which of the following?
(A) Severe chronic suppurative keratitis with ulceration
(B) Complete anterior lens luxation with corneal contact
(C) Retinal ganglion cell loss and optic disc cupping
(D) Marked hypertrophy of the secretory ciliary body

2012

25. Consider the following statements regarding epidermal responses to injury:
I. Hyperkeratosis is an increased thickness of the stratum corneum.
II. Parakeratosis is characterized by the retention of nuclei within the cells of the stratum corneum.
III. Acanthosis refers to intercellular edema within the epidermal layers.
Which of the above statements are correct?
(A) I and II
(B) II and III
(C) I and III
(D) III only

Downloaded from www.pdf.tube

2005

81. In the pathological mechanism of abortion due to Toxoplasma gondii in sheep, the most characteristic gross lesion is found on the placenta. Which of the following describes this lesion?
(A) Leathery, thickened intercotyledonary areas
(B) Dark red, necrotic cotyledons with characteristic white calcified foci
(C) Bright red, swollen cotyledons with no exudate
(D) Diffuse edema of the chorioallantois without specific cotyledonary damage

2008

82. Which of the following classifications of mastitis is characterized by hard, fibrotic quarters, often with microabscesses, and is typical of chronic Staphylococcus aureus infection?
(A) Acute catarrhal to necrotizing mastitis
(B) Acute gangrenous and hemorrhagic mastitis
(C) Fibrinous to fibrinosuppurative mastitis
(D) Suppurative to granulomatous mastitis

2017

83. Epididymitis in rams is a significant cause of infertility and economic loss. Which infectious agent is the most common cause of contagious epididymitis in sheep?
(A) Brucella ovis
(B) Campylobacter fetus
(C) Corynebacterium renale
(D) Tritrichomonas foetus

2006

84. Orchitis is inflammation of the testis. In severe cases of purulent orchitis, what secondary pathological change inevitably leads to permanent sterility in the affected testis?
(A) Squamous metaplasia of the sustentacular Sertoli cells
(B) Malignant neoplastic transformation into a seminoma
(C) Tubular necrosis and subsequent interstitial fibrosis
(D) Diffuse hyperplasia of the interstitial Leydig cells

2013

85. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).
Assertion (A): Astrogliosis is a delayed but permanent response to central nervous system injury, often resulting in a "glial scar."
Reason (R): Astrocytes rapidly proliferate and deposit large amounts of collagen to bridge the defect in the brain parenchyma.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2021

86. Hydrocephalus is characterized by an abnormal accumulation of cerebrospinal fluid. Non-communicating (obstructive) hydrocephalus is most frequently caused by an anomaly or obstruction at which anatomical site?
(A) Foramen of Monro
(B) Lateral apertures (Foramina of Luschka)
(C) Arachnoid villi
(D) Mesencephalic aqueduct (Aqueduct of Sylvius)

2010

87. Polioencephalomalacia (PEM) in ruminants causes cerebrocortical necrosis. What specific clinical diagnostic phenomenon is observed when fresh brain tissue from a PEM case is exposed to ultraviolet light?
(A) Complete absorption of UV light
(B) Brilliant autofluorescence
(C) Rapid photobleaching
(D) Production of a distinct odor

2002

88. Osteopetrosis is a congenital bone disease characterized by increased bone density but extreme fragility. This pathology results from a functional defect in which specific cell type?
(A) Osteoblasts
(B) Osteocytes
(C) Osteoclasts
(D) Chondrocytes

2022

89. Suppurative arthritis in neonatal farm animals is most commonly a sequela to which of the following?
(A) Direct penetrating trauma to the joint capsule
(B) Extension of infection from adjacent osteomyelitis
(C) Autoimmune targeting of the synovial membrane
(D) Hematogenous spread from omphalophlebitis or enteritis

NODIA

(C) It contains the highest concentration of cytochrome P450 enzymes.
(D) It serves as the primary metabolic site of bile acid synthesis.

2021
72. Cholangiohepatitis involves inflammation of the biliary tract extending into the hepatic parenchyma. In cats, this condition is often seen concurrently with inflammation of which two other organs (triaditis)?
(A) Stomach and small intestine
(B) Kidney and spleen
(C) Pancreas and duodenum
(D) Gallbladder and right kidney

2003
73. Chronic pancreatitis eventually leads to exocrine pancreatic insufficiency. Morphologically, a chronically inflamed pancreas is typically characterized by which set of features?
(A) Edematous, hemorrhagic, and enlarged
(B) Cystic, massively enlarged, and fluctuant
(C) Diffusely pale, friable, and swollen
(D) Firm, nodular, shrunken, and fibrotic

2010
74. The fundamental mechanism of renal failure due to acute tubular necrosis involves anuria or oliguria. Which of the following is a primary contributor to the reduced glomerular filtration rate in these cases?
(A) Tubular obstruction by cellular casts
(B) Dilation of the afferent arterioles
(C) Hyperplasia of the macula densa cells
(D) Increased renal medullary blood flow

2012
75. Consider the following statements regarding interstitial nephritis:
I. Interstitial nephritis primarily targets the renal interstitium and tubules.
II. Leptospirosis is a classic bacterial cause of interstitial nephritis in dogs.
III. White spotted kidneys in calves are an example of chronic interstitial nephritis.
IV. Glomeruli are the primary initial target in interstitial nephritis.
Which of the above statements is/are incorrect?
(A) I and II
(B) III and IV
(C) I, II and III
(D) IV only

2010
76. Ascending pyelonephritis requires the movement of bacteria from the lower urinary tract to the kidneys. Which of the following anatomical defenses must be overcome or compromised for this to occur?
(A) The external urethral sphincter
(B) The vesicoureteral valve mechanism
(C) The renal capsule
(D) The glomerular basement membrane

2013
77. Feline lower urinary tract disease (FLUTD) often presents with urethral obstruction in male cats. What is the typical composition of the urethral plug in these cases?
(A) Struvite crystals and proteinaceous matrix
(B) Solid, large calcium oxalate mineral calculi
(C) Pure urate sand mixed with inflammatory cells
(D) Desquamated transitional epithelial cells only

2018
78. Morphological types of cystitis include follicular cystitis. What is the distinguishing gross pathological feature of follicular cystitis?
(A) Severe mucosal hemorrhage with blood clots in the lumen
(B) Multiple gas-filled vesicles within the bladder wall
(C) Small, raised, gray-white mucosal lymphoid nodules
(D) Diffuse mucosal ulceration with diphtheritic membranes

2023
79. Cystic ovarian disease in dairy cattle most commonly involves follicular cysts. These cysts arise due to a failure of ovulation, which is fundamentally caused by a lack of which reproductive hormone surge?
(A) Progesterone
(B) Estrogen
(C) Prostaglandin F_2 alpha
(D) Luteinizing Hormone (LH)

2003
80. Endometritis is inflammation limited to the uterine mucosa. In mares, chronic endometritis frequently causes infertility due to which microscopic change?
(A) Squamous metaplasia of the epithelium
(B) Diffuse necrosis of the myometrium
(C) Periglandular fibrosis
(D) Endometrial ossification

Click Here to Buy book on Amazon

disease

2015

99. Uremic pericarditis is a classical lesion observed in severe chronic renal failure. The morphological pattern of this pericarditis is primarily:
(A) Fibrinous
(B) Purulent
(C) Granulomatous
(D) Serous

2012

100. Arteritis can be induced by viral infections. Equine Viral Arteritis (EVA) primarily targets which layer of the blood vessel wall, leading to edema and hemorrhage?
(A) Tunica intima
(B) Tunica media
(C) Tunica adventitia
(D) Vasa vasorum only

2013

101. Lymphomas are classified by their anatomical distribution. Which form of lymphoma is characterized by primary involvement of the thymus, mediastinal lymph nodes, and potentially causing chylothorax in cats?
(A) Mediastinal form
(B) Multicentric form
(C) Alimentary form
(D) Cutaneous form

2005

102. Myeloproliferative disorders represent neoplastic proliferations of non-lymphoid hematopoietic cells in the bone marrow. Which of the following is a classic characteristic of a myeloid leukemia?
(A) Predominance of differentiated plasma cells within the bone marrow
(B) Infiltration of solid organs exclusively by mature T-lymphocytes
(C) Complete suppression of all active hematopoietic cell lineages
(D) Massive proliferation of non-lymphoid precursors in marrow and blood

2007

103. Rhinitis can be classified by its exudate. Catarrhal rhinitis is characterized by the hypersecretion of mucus and is most commonly the initial stage of which type of respiratory infection?
(A) Severe invasive fungal infections
(B) Mild viral infections
(C) Chronic granulomatous diseases
(D) Parasitic migrations

2002

104. Laryngeal hemiplegia (roaring) in horses is a structural airway obstruction caused by atrophy of the cricoarytenoideus dorsalis muscle. This atrophy is neurogenic, primarily due to degeneration of which nerve?
(A) Right recurrent laryngeal nerve
(B) Glossopharyngeal nerve
(C) Left recurrent laryngeal nerve
(D) Vagus nerve trunk

2011

105. Consider the following statements regarding the pathogenesis of pneumonias:
I. Bronchopneumonia characteristically originates at the bronchiolar-alveolar junction.
II. Interstitial pneumonia is usually caused by inhaled bacteria.
III. Fibrinous pneumonia involves severe vascular damage and fluid leakage.
Which of the above statements are correct?
(A) I and III
(B) I and II
(C) II and III
(D) II only

NODIA

2018

106. In the classification of pneumonias, aspiration pneumonia typically results in a severe necrotizing or gangrenous lesion. This specific severity is primarily due to which combination of factors?
(A) Gastric acid irritation and mixed bacterial infection
(B) Autoimmune response to inhaled food particles and proteins
(C) Direct vascular thrombosis and ischemia of alveolar tissue
(D) Viral replication and proliferation within aspirated fluid

2002

107. Pulmonary edema can be cardiogenic or non-cardiogenic. Which of the following is a classic mechanism for non-cardiogenic pulmonary edema?
(A) Increased pulmonary capillary hydrostatic pressure
(B) Increased alveolar capillary permeability
(C) Decreased plasma oncotic pressure within systemic vessels
(D) Obstruction of lymphatic drainage in pulmonary tissue

2015

125. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).

Assertion (A): Coliform mastitis in dairy cattle frequently leads to severe systemic illness, shock, and sometimes death.

Reason (R): Coliform bacteria produce potent endotoxins that cause direct necrosis of the mammary secretory epithelium.

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2010

126. Epididymitis is frequently accompanied by peritesticular changes. In severe chronic epididymitis, a common consequence that physically restricts the testis and impairs thermoregulation is which of the following?

(A) Excessive accumulation of fluid in the vaginal cavity
(B) Prominent dilation of the pampiniform plexus veins
(C) Fibrous adhesions of the vaginal tunics
(D) Acute rotational torsion of the spermatic cord

2023

127. Microgliosis is a key response to neural injury. When microglia become phagocytic and engorged with lipid debris from necrotic brain tissue, they are specifically referred to as which of the following?

(A) Gemistocytes
(B) Spheroids
(C) Rosenthal fibers
(D) Gitter cells

2004

128. Spongiform encephalopathies are characterized by vacuolation. In Bovine Spongiform Encephalopathy (BSE), the characteristic vacuoles are predominantly located in which anatomical region of the central nervous system?

(A) Gray matter of the cerebral cortex
(B) Astrocytes of cerebellar white matter
(C) Neurons of the brainstem obex
(D) Sensory tract of spinal cord dorsal horns

2002

129. Rickets is a disease of growing bone. Which of the following best describes the cardinal gross lesion seen at the costochondral junctions of animals with rickets?

(A) Thinning and extreme fragility of the ribs
(B) Nodular 'rachitic rosary' enlargements
(C) Complete fusion of the costochondral junction
(D) Suppurative exudate tracking along the rib shafts

2012

130. Arthritis can be classified by its exudate. Erysipelothrix rhusiopathiae infection in swine characteristically produces which morphological type of chronic arthritis?

(A) Acute suppurative or purulent arthritis
(B) Severe necrotizing hemorrhagic arthritis
(C) Chronic lymphocytic or serous arthritis
(D) Fibrinous-proliferative arthritis

2014

131. Myositis can be eosinophilic in nature. Eosinophilic myositis in dogs is a specific immune-mediated disease targeting the muscles of mastication. What specific anatomical feature of these muscles makes them the target?

(A) They express a unique Type 2M myosin isoform.
(B) They lack a protective fascial sheath around myofibers.
(C) They rely exclusively on active anaerobic glycolysis.
(D) They contain very high concentrations of muscle myoglobin.

2003

132. Primary hypothyroidism in dogs is most commonly caused by which of the following pathological processes?

(A) Chronic dietary iodine deficiency
(B) Metastatic thyroid follicular carcinoma
(C) Lymphocytic thyroiditis
(D) Pituitary or hypothalamic dysfunction

2012

133. Hyperparathyroidism secondary to chronic renal failure is caused by a complex mechanism. Which specific endocrine failure of the diseased kidney is a direct catalyst for decreased intestinal calcium absorption and subsequent parathyroid stimulation?

(A) Decreased calcitriol synthesis
(B) Decreased erythropoietin production
(C) Increased renal synthesis of renin
(D) Decreased parathyroid hormone clearance

Downloaded from www.pdf.tube

(D) I and IV

2025
116. In foundational renal pathology, what is the term used to describe the primary morphological unit of injury involving both the glomerulus and its associated tubule, reflecting the fact that damage to one inevitably affects the other?
(A) Renal corpuscle
(B) Malpighian pyramid
(C) Nephron
(D) Juxtaglomerular apparatus

2004
117. Toxic nephropathy specifically targets the proximal convoluted tubules. Which of the following is a classic nephrotoxin causing acute tubular necrosis characterized by oxalate crystal deposition within the tubular lumens?
(A) Ethylene glycol
(B) Aminoglycoside antibiotics
(C) Lead
(D) Non-steroidal anti-inflammatory drugs

2022
118. Glomerulonephritis is morphologically classified by the pattern of cellular proliferation and membrane changes. Which type is characterized by marked thickening of the capillary basement membranes without significant cellular proliferation, commonly leading to nephrotic syndrome?
(A) Proliferative glomerulonephritis
(B) Membranoproliferative glomerulonephritis
(C) Glomerulosclerosis
(D) Membranous glomerulonephritis

2015
119. Ascending pyelonephritis typically features distinct macroscopic lesions. Which of the following best describes the classical gross appearance of acute pyelonephritis in a kidney?
(A) Diffusely smooth, pale, and symmetrically enlarged cortical parenchyma
(B) Purulent exudate in the pelvis radiating into the parenchyma
(C) Irregular, depressed cortical scars from previous infarcts
(D) Multiple large, clear fluid-filled cysts within the cortex

2024
120. Urolithiasis formation involves complex physical and chemical factors. A fundamental prerequisite for the precipitation of any mineral forming a urolith is which of the following?
(A) Complete structural obstruction of distal urine flow
(B) A persistent and strictly acidic urinary pH level
(C) Supersaturation of urine with the crystalloid
(D) The persistent presence of a central bacterial nidus

NODIA

2025
121. In cases of chronic cystitis, the mucosa may undergo metaplastic changes. What is the specific term for the mucosal response characterized by the formation of multiple fluid-filled cystic structures projecting into the bladder lumen?
(A) Polypoid cystitis
(B) Follicular cystitis
(C) Emphysematous cystitis
(D) Cystitis cystica

2007
122. Cystic ovarian disease in swine is often associated with reproductive failure. Multiple large, thick-walled cysts producing excess progesterone and causing anestrus are classified as which of the following?
(A) Luteal cysts
(B) Follicular cysts
(C) Cystic corpora lutea
(D) Parovarian cysts

2003
123. The pyometra complex in dogs is fundamentally an endocrine-driven disease secondarily complicated by bacteria. Which bacterium is the most frequently isolated pathogen in canine pyometra?
(A) Staphylococcus pseudintermedius
(B) Escherichia coli
(C) Streptococcus canis
(D) Brucella canis

2003
124. Pathological mechanisms of abortion can be classified by the timing and nature of fetal death. Which infectious agent is classically associated with early embryonic or fetal death in cattle, where the dead fetus is sometimes retained and macerated, leading to post-coital pyometra?
(A) Campylobacter fetus subspecies
(B) Tritrichomonas foetus
(C) Neospora caninum
(D) Bovine Herpesvirus-1

lymph node
(D) Lymphadenitis – Inflammation of the lymph node

2022

144. Thymic hypoplasia is a primary immunodeficiency. In Arabian foals, severe combined immunodeficiency (SCID) is an autosomal recessive trait characterized by a profound lack of which cell populations?
(A) Neutrophils and macrophages
(B) Erythrocytes and platelets
(C) B and T lymphocytes
(D) Eosinophils and basophils

2004

145. Consider the following statements regarding the pathology of the upper respiratory tract:
I. Rhinitis can be classified as catarrhal, fibrinous, purulent, or granulomatous.
II. Sinusitis is rarely accompanied by rhinitis.
III. Tracheitis in dogs is a common feature of the canine infectious respiratory disease complex.
IV. Structural airway obstructions like laryngeal paralysis primarily affect expiration.
V. Guttural pouch empyema in horses is a specific form of upper airway pathology.
Which of the above statements are INCORRECT?
(A) I, III and V
(B) II, IV and V
(C) II and IV
(D) I and III

2024

146. Bronchopneumonia is predominantly caused by bacteria. The initial deposition of these inhaled bacteria primarily occurs at which specific anatomical site in the respiratory tract?
(A) Mucosa of the nasal cavity
(B) Bifurcation of the main trachea
(C) Mesothelial visceral pleura
(D) Bronchioloalveolar junction

2007

147. Atelectasis refers to the incomplete expansion of the lungs or the collapse of previously inflated lungs. Which type of atelectasis is typically observed in animals that are bedridden or recumbent for prolonged periods?
(A) Hypostatic atelectasis
(B) Obstructive atelectasis
(C) Compressive atelectasis
(D) Congenital atelectasis

2016

148. Vesicular stomatitis presents with vesicles and bullae in the oral cavity. Microscopically, the initial formation of these vesicles occurs via which pathomechanism?
(A) Massive influx of neutrophils causing focal microabscesses
(B) Necrosis of the basal cell layer leading to subepidermal clefts
(C) Spongiosis and cell ballooning in the stratum spinosum
(D) Direct rupture of intraepithelial capillary blood vessels

2017

149. In the pathology of the ruminant forestomach, primary ruminal tympany (frothy bloat) is primarily driven by the ingestion of certain legumes. The frothy nature of the rumen contents is stabilized by which specific plant proteins?
(A) Soluble leaf proteins
(B) Insoluble plant lignins
(C) Condensation products of tannins
(D) Plant cellulose and hemicellulose

2010

150. Arrange the following events in the pathogenesis of left displaced abomasum (LDA) in chronological order:
I. Accumulation of gas in the abomasum
II. Displacement of the abomasum under the rumen to the left abdominal wall
III. Abomasal hypomotility (atony)
IV. Partial obstruction of abomasal outflow
(A) III, I, II, IV
(B) I, III, II, IV
(C) III, II, I, IV
(D) II, I, IV, III

2013

151. Gastric ulcers in dogs are often associated with the administration of non-steroidal anti-inflammatory drugs (NSAIDs). The primary mechanism for NSAID-induced gastric ulceration is the inhibition of which enzyme?
(A) Lipoxygenase
(B) Cyclooxygenase
(C) Phospholipase A_2
(D) Thromboxane synthase

2003

152. Hepatic injury often results in the leakage of intracellular enzymes into the blood. Which of the following is an identifying correct statement regarding hepatic enzymes in small animals?
(A) AST is exclusively found in liver tissue of

Click Here to Buy book on Flipkart

2002

134. Adrenal cortical disorders include hyperadrenocorticism. In naturally occurring canine hyperadrenocorticism, the most frequent underlying etiology is which of the following?
(A) Functional adrenocortical carcinoma
(B) Functional adrenocortical adenoma
(C) Ectopic ACTH secretion by a non-endocrine tumor
(D) Functional pituitary adenoma secreting ACTH

2001

135. Match the endocrine pathology in List I with its classic clinical or pathological feature in List II.
List I: Endocrine Pathology
P. Diabetes mellitus
Q. Hypoadrenocorticism
R. Hyperthyroidism
S. Pituitary Pars Intermedia Dysfunction (PPID)
List II: Clinical/Pathological Feature
1. Hyperkalemia and hyponatremia
2. Hirsutism (hypertrichosis)
3. Polyuria, polydipsia, and glucosuria
4. Weight loss despite polyphagia in older cats
(A) P-3, Q-1, R-4, S-2
(B) P-4, Q-3, R-1, S-2
(C) P-3, Q-2, R-4, S-1
(D) P-1, Q-4, R-2, S-3

2008

136. Otitis externa in dogs is frequently complicated by secondary overgrowth of commensal organisms. Which characteristic yeast with a "peanut" or "footprint" shape is commonly identified cytologically in these cases?
(A) Candida albicans pseudohyphae
(B) Cryptococcus neoformans yeast
(C) Malassezia pachydermatis
(D) Histoplasma capsulatum

2013

137. Which of the following ophthalmic pathologies is defined as inflammation of the uveal tract (iris, ciliary body, and choroid)?
(A) Keratitis
(B) Conjunctivitis
(C) Blepharitis
(D) Uveitis

2017

138. Which of the following dermatological terms refers to an increased thickness of the stratum spinosum of the epidermis, typically resulting from chronic irritation?
(A) Acanthosis
(B) Hyperkeratosis
(C) Dyskeratosis
(D) Spongiosis

2005

139. A pustule is a classic morphological pattern of dermatitis. In dogs, superficial pustular dermatitis is most commonly caused by which bacterial pathogen?
(A) Multi-resistant Pseudomonas aeruginosa
(B) Staphylococcus pseudintermedius
(C) Beta-hemolytic Streptococcus pyogenes
(D) Atypical Mycobacterium fortuitum

2011

140. In evaluating congenital heart defects, which condition consists of a high ventricular septal defect, dextroposition of the aorta, pulmonic stenosis, and right ventricular hypertrophy?
(A) Eisenmenger complex physiology
(B) Tetralogy of Fallot
(C) Cor triatriatum
(D) Truncus arteriosus

2004

141. Pathological alterations of the myocardium include nutritional myopathy. While skeletal muscle is widely affected, myocardial involvement in calves and lambs classically presents as pale plaques primarily located in which specific cardiac region?
(A) Left ventricular subendocardium
(B) Myocardium of the left atrium
(C) Subepicardial zone of the right ventricle
(D) Connective tissue of the interatrial septum

2011

142. Aneurysms are rare but can be lethal if they rupture. In pigs, a potentially fatal dissecting aneurysm of the aorta is a known complication of deficiency in which essential trace mineral?
(A) Zinc
(B) Iron
(C) Copper
(D) Selenium

2013

143. Which of the following is an example of an identifying correct pair regarding lymph node pathology?
(A) Lymphadenopathy – Specific benign neoplasia of the lymph node
(B) Lymphangitis – Neoplastic infiltration of the lymph node
(C) Lymphoma – Acute bacterial infection of the

Downloaded from www.pdf.tube

2022

162. Basic cellular responses to neural injury include Wallerian degeneration. After axonal transection, the proximal stump of the axon undergoes swelling and attempts to regenerate. What is this regenerating tip called?
(A) Spheroid
(B) Axonal torpedo
(C) Neurofibrillary tangle
(D) Growth cone

2019

163. Viral encephalitis often targets specific cell types. In canine distemper virus infection, the virus targets which glial cell, leading to primary demyelination in the central nervous system?
(A) Oligodendrocytes
(B) Reactive astrocytes
(C) Phagocytic microglia
(D) Ependymal cells of ventricles

2013

164. Spongiform encephalopathies are associated with the accumulation of an abnormal protein. The pathogenic prion protein (PrPSc) differs from the normal cellular prion protein (PrPC) primarily in which aspect?
(A) Primary amino acid sequence of the protein
(B) Post-translational addition of a glycolipid anchor
(C) Three-dimensional conformational structure
(D) Specific genomic location of the PRNP gene locus

2013

165. Given below are two statements, one labelled as Assertion (A) and the other labelled as Reason (R).
Assertion (A): Polioencephalomalacia in sheep and cattle can be successfully treated with early intravenous administration of thiamine.
Reason (R): Rumen microbes natively produce thiaminases that directly destroy thiamine before it can be absorbed.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2015

166. Metabolic bone diseases include osteomalacia. Which of the following is an identifying correct statement regarding osteomalacia?
(A) It involves decreased bone mass but normal mineralization.
(B) It primarily affects the growth plates of long bones.
(C) It is characterized by excessive and completely haphazard woven bone formation.
(D) It involves the accumulation of unmineralized osteoid in adult bone.

2019

167. Pathological changes in skeletal muscle include nutritional myopathy (White Muscle Disease). The severity of the lesions depends on the muscle's activity level. In calves, which muscles are typically most severely affected?
(A) Cranial facial muscles
(B) Highly active skeletal muscles
(C) Deep extraocular muscles
(D) Gastrointestinal smooth muscles

2005

168. Goiter is defined as non-neoplastic, non-inflammatory enlargement of the thyroid gland. In neonates born to dams with iodine deficiency, the most severe consequence of congenital hypothyroidism is:
(A) Accelerated skeletal growth
(B) Hyperactivity and tachycardia
(C) Prolonged gestation and alopecia
(D) Severe and immediate postnatal hyperthermia

2024

169. Primary hyperparathyroidism leads to severe metabolic disturbances. Which set of serum biochemical abnormalities is the hallmark of primary hyperparathyroidism?
(A) Hypocalcemia and hyperphosphatemia
(B) Hypercalcemia and hyperphosphatemia
(C) Hypocalcemia and hypophosphatemia
(D) Hypercalcemia and hypophosphatemia

NODIA

2007

170. Adrenal pathology includes hypoadrenocorticism. The classic electrolyte imbalance seen in Addison's disease (hyponatremia and hyperkalemia) is directly attributable to the loss of which specific hormone?
(A) Aldosterone
(B) Cortisol
(C) Epinephrine
(D) Dehydroepiandrosterone (DHEA)

2011

171. In cases of chronic diabetes mellitus, domestic animals (especially dogs) are highly prone to

small animals.
(B) ALP is a primary marker for acute hepatocellular necrosis.
(C) ALT is specific for hepatocyte injury in dogs and cats.
(D) GGT is typically elevated in cases of skeletal muscle trauma.

2001

153. Hepatic cirrhosis involves extensive remodeling. The primary cell type responsible for the deposition of collagen (fibrosis) in the perisinusoidal space of Disse during chronic liver injury is the:
(A) Parenchymal hepatocyte
(B) Macrophage Kupffer cell
(C) Sinusoidal endothelial cell
(D) Hepatic stellate cell

2010

154. Exocrine pancreatic insufficiency (EPI) causes specific clinical signs. Which of the following tests is considered the "gold standard" diagnostic assay for detecting EPI in dogs?
(A) Serum TLI
(B) Serum amylase
(C) Serum lipase
(D) Fecal elastase

2011

155. Acute tubular necrosis (ATN) can be distinguished by the condition of the tubular basement membrane (TBM). If the TBM is completely destroyed (tubulorrhexis), what is the expected outcome of the tubular epithelium?
(A) Rapid, perfect regeneration
(B) Imperfect regeneration with interstitial fibrosis
(C) Regeneration accompanied by multinucleated giant cells
(D) Immediate malignant transformation

2017

156. In the pathogenesis of glomerulonephritis, immune complex deposition leads to complement activation. Which specific component of the complement cascade is a potent chemotactic factor that draws neutrophils into the glomerulus, exacerbating the damage?
(A) C1q complex
(B) C5a
(C) C3b molecule
(D) C9 terminal

2024

157. Feline lower urinary tract disease (FLUTD) represents a complex of disorders. In cases designated as feline idiopathic cystitis (FIC), which of the following mechanisms is strongly suspected to play a central role?
(A) Neurogenic inflammation related to stress
(B) Chronic viral infection by Calicivirus
(C) Autoimmune attack on the detrusor muscle
(D) Primary anatomical stricture of the urethra

2008

158. Female reproductive pathology includes pyometra. While dogs typically develop pyometra under progesterone dominance, in cattle, pyometra is almost exclusively associated with the retention of which structure on the ovary?
(A) Follicular cyst
(B) Granulosa cell tumor
(C) Corpus luteum
(D) Luteinized cyst

2004

159. Metritis and endometritis are distinct conditions. Which of the following represents the most accurate definition of metritis?
(A) Inflammation limited to the superficial layers of the endometrium
(B) Accumulation of sterile mucoid fluid within the uterine lumen
(C) Neoplastic proliferation of endometrial and glandular epithelial cells
(D) Inflammation of all layers of the uterine wall

2007

160. Mastitis classifications include subclinical mastitis, which causes significant economic loss. What is the primary indicator used to detect subclinical mastitis in a dairy herd?
(A) Presence of clots in the milk
(B) Swelling and redness of the udder tissue
(C) Elevated somatic cell count
(D) Moderate systemic fever in the cow

2025

161. In male reproductive pathology, benign prostatic hyperplasia (BPH) commonly occurs in older intact dogs. What is a frequent macroscopic complication of BPH that can cause significant clinical signs such as tenesmus?
(A) Malignant transformation into prostatic adenocarcinoma
(B) Prostatic cyst formation and abscessation
(C) Calcification of the vas deferens
(D) Atrophy of the bulbo-urethral glands

Click Here to Buy book on Amazon

NODIA

2013

108. Ruminal tympany (bloat) results in a specific gross pathological lesion due to extreme distension of the rumen compressing the diaphragm and thoracic vessels. Which of the following is the pathognomonic lesion observed at necropsy?
(A) "Bread and butter" pericarditis of the heart
(B) "Bloat line" along the esophageal mucosa
(C) Diffuse petechiation of the renal cortex
(D) Extensive fatty change of the hepatic parenchyma

2006

109. Right displaced abomasum (RDA) is considered a more severe and acute medical emergency than LDA because RDA frequently progresses to which of the following complications?
(A) Abomasal volvulus leading to ischemia and necrosis
(B) Direct rupture into the peritoneal cavity
(C) Immediate development of a perforated gastric ulcer
(D) Prolapse of the abomasum through the diaphragm

2021

110. Which of the following represents the correct sequence of pathophysiological events in acute grain engorgement leading to ruminal acidosis?
(A) Grain ingestion → Proliferation of Streptococcus bovis → Fall in ruminal pH → Proliferation of Lactobacilli → Lactic acidosis
(B) Grain ingestion → Proliferation of Lactobacilli → Rise in ruminal pH → Proliferation of Streptococcus bovis → Lactic acidosis
(C) Grain ingestion → Fall in ruminal pH → Proliferation of protozoa → VFA production → Lactic acidosis
(D) Grain ingestion → Proliferation of Fibrobacter → Lactic acid utilization → Acidosis → Death

2003

111. Specific enteritis classifications include granulomatous enteritis. Which of the following diseases is the classic example of granulomatous enteritis in cattle?
(A) Winter Dysentery
(B) Bovine Viral Diarrhea
(C) Salmonellosis
(D) Johne's Disease

2014

112. Hepatic lipidosis (fatty liver) occurs when the rate of hepatic triglyceride accumulation exceeds the rate of metabolic degradation and release. In dairy cattle, this condition is most closely associated with which metabolic state?
(A) Positive energy balance during the late lactation period
(B) High dietary intake of bypass proteins and soluble carbohydrates
(C) Negative energy balance during the periparturient period
(D) Chronic copper accumulation leading to hepatocyte necrosis

2011

113. Cirrhosis is the end-stage of chronic hepatic injury. Which of the following functional consequences directly results from the severe fibrosis and architectural distortion characteristic of cirrhosis?
(A) Increased synthesis of albumin by active hepatocytes
(B) Portal hypertension and acquired portosystemic shunts
(C) Hypoglycemia secondary to persistent hyperinsulinemia
(D) Excessive storage of glycogen within hepatic parenchyma

2021

114. Cholangitis is defined as inflammation of the biliary ducts. Liver fluke infection (Fasciola hepatica) in ruminants classically causes which specific type of chronic biliary pathology?
(A) Suppurative cholangitis
(B) Lymphoplasmacytic cholangitis
(C) Fibrinonecrotic cholangitis
(D) Chronic fibrosing cholangitis

2014

115. Consider the following statements regarding exocrine pancreatic pathology:
I. Acute pancreatitis frequently involves enzymatic fat necrosis.
II. Chronic pancreatitis is a common cause of exocrine pancreatic insufficiency in dogs.
III. Exocrine pancreatic insufficiency leads to profound maldigestion and steatorrhea.
IV. Trypsinogen activation is the protective mechanism against acute pancreatitis.
Which of the above statements are correct?
(A) I, II and III
(B) I, III and IV
(C) II, III and IV

developing which of the following specific ocular pathologies?
(A) Anterior uveitis
(B) Bilateral cataracts
(C) Retinal detachment
(D) Squamous cell carcinoma of the conjunctiva

2004

172. Otitis media in dogs and cats frequently occurs as a direct extension of infection from which primary source?
(A) Hematogenous spread from a chronic systemic bacteremia
(B) Extension from chronic otitis externa
(C) Ascending infection from pharynx via the auditory tube
(D) Direct penetrating trauma through the petrous temporal bone

2009

173. Dermatological pathology features distinct patterns. Which of the following defines parakeratosis?
(A) Stratum corneum thickening with nuclear retention
(B) Significantly increased pigmentation of the epidermal basal layer
(C) Loss of intercellular bridges within the stratum spinosum layer
(D) Marked accumulation of serous edema fluid within the dermis

2024

174. The major morphological pattern of dermatitis characterized by prominent intraepidermal vesicles or bullae, often seen in immune-mediated conditions like pemphigus vulgaris, is known as:
(A) Superficial perivascular dermatitis
(B) Deep nodular dermatitis
(C) Vesiculopustular dermatitis
(D) Interface or lichenoid dermatitis

2016

175. Match the gastrointestinal pathology in List I with its primary anatomical location in List II.
List I: Gastrointestinal Pathology
P. Abomasal displacement
Q. Ruminal acidosis
R. Johne's disease
S. Proliferative hemorrhagic enteropathy
List II: Anatomical Location
1. Ileum
2. Forestomach
3. Glandular stomach of ruminants
4. Terminal ileum, cecum, and proximal colon

(A) P-3, Q-2, R-4, S-1
(B) P-2, Q-3, R-1, S-4
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-2, S-3

2013

176. Congenital heart defects also include Ventricular Septal Defects (VSD). In domestic animals, the most common anatomical location for a VSD is in which portion of the septum?
(A) Muscular portion near the apex of the heart
(B) Within the muscular interatrial septum
(C) High membranous portion
(D) At the apex region of the right ventricle

2002

177. Myocarditis can be caused by parasites. In dogs, sudden death due to severe multifocal necrotizing myocarditis is a known complication of infection by which protozoan parasite?
(A) Toxoplasma gondii oocysts
(B) Trypanosoma cruzi
(C) Neospora caninum
(D) Babesia canis

2023

178. Thrombosis involves the formation of a clot within the vasculature. The classical appearance of an arterial thrombus, characterized by alternating layers of platelets/fibrin and red blood cells, is referred to as:
(A) Virchow's nodes
(B) Aschoff bodies
(C) Negri bodies
(D) Lines of Zahn

2008

179. Lymphomas can be histologically graded. A lymphoma characterized by small, well-differentiated lymphocytes with a low mitotic index is generally classified as:
(A) Low-grade
(B) High-grade
(C) Intermediate-grade
(D) Anaplastic

2023

180. Bone marrow aplasia (aplastic anemia) can result from idiosyncratic drug reactions. Which of the following antibiotics is classically associated with inducing fatal aplastic anemia in humans and occasionally animals, leading to restricted use in food animals?
(A) Penicillin G sodium
(B) Oxytetracycline
(C) Chloramphenicol

Downloaded from www.pdf.tube

(D) Enrofloxacin solution

2024

181. Rhinitis in swine is economically important. Inclusion body rhinitis, causing severe disease primarily in young piglets, is caused by which type of virus?
(A) Porcine Cytomegalovirus
(B) Porcine Circovirus
(C) Swine Influenza Type A Virus
(D) Porcine Parvovirus

2018

182. Bronchopneumonia is morphologically characterized by a specific distribution of lesions. Why are the cranioventral lung lobes overwhelmingly the most affected region in bacterial bronchopneumonia in quadrupeds?
(A) They have the poorest vascular blood supply in the lungs.
(B) They completely lack resident alveolar macrophages.
(C) They are directly exposed to inspired cold environmental air.
(D) Gravitational settling and short, direct airways

2015

183. Emphysema in animals is most commonly a secondary lesion. In cattle, acute interstitial emphysema and edema (fog fever) occurs following the abrupt introduction to lush pasture due to the ruminal conversion of L-tryptophan to which pneumotoxic compound?
(A) Ammonia
(B) 3-methylindole
(C) Hydrogen sulfide
(D) Lactic acid

2013

184. In vesicular stomatitis, the presence of intact vesicles is rare by the time of clinical examination because they rapidly rupture. What is the characteristic gross lesion left immediately after a vesicle ruptures?
(A) A deep, actively bleeding ulcer
(B) A raised, proliferative chronic granuloma
(C) A shallow, ragged erosion
(D) A firm, dense fibrous scar tissue

2002

185. Consider the following statements regarding gastric and intestinal pathologies:
I. Right displaced abomasum (RDA) is less likely to progress to volvulus than LDA.
II. Gastric ulcers in pigs are primarily found in the pars esophagea.
III. Johne's disease causes a protein-losing enteropathy.
IV. Parvoviral enteritis in dogs targets the crypt epithelium.
V. Salmonella primarily induces a catarrhal enteritis with minimal tissue destruction.
Which of the above statements are correct?
(A) II, III and IV
(B) I, II and III
(C) III, IV and V
(D) II, IV and V

2024

186. Hepating necrosis patterns provide diagnostic clues. Which pattern of necrosis is most characteristic of acute toxic insult from agents like pyrrolizidine alkaloids or aflatoxins in their late stages, often bridging adjacent central veins?
(A) Massive bridging necrosis
(B) Focal multifocal necrosis
(C) Strictly periportal zone necrosis
(D) Paracentral lobular necrosis

2017

187. Exocrine pancreatic insufficiency (EPI) leads to a failure of digestion. Which of the following is an identifying correct statement regarding the clinical presentation of a dog with severe EPI?
(A) Rapid body weight gain despite severe anorexia
(B) Steatorrhea with ravenous appetite
(C) Severe colonic constipation and tenesmus
(D) Dark, tarry black stools from melena

NODIA

2013

188. In evaluating renal failure mechanisms, the term "uremia" refers to the clinical syndrome of renal failure. Which of the following extrarenal lesions is classically associated with uremia?
(A) Mucocele formation in the gallbladder
(B) Ulcerative glossitis and stomatitis
(C) Rupture of the cranial cruciate ligament
(D) Hyperplasia of the thyroid gland

2018

189. Glomerulonephritis frequently results in significant protein loss in the urine. The loss of which specific plasma protein is primarily responsible for the development of systemic edema and ascites in nephrotic syndrome?
(A) Albumin
(B) Fibrinogen
(C) Globulins
(D) Prothrombin

2009

190. Feline lower urinary tract disease (FLUTD) can result in post-renal azotemia. If a urethral obstruction is not relieved, death will typically occur within days primarily due to which specific biochemical derangement?
(A) Severe hypocalcemia
(B) Profound hypernatremia
(C) Fatal hyperkalemia
(D) Acute hypoglycemia

2017

191. In female reproductive pathology, the term "pyometra" refers to an infected, pus-filled uterus. In bitches, the classical timing for the development of pyometra is during which phase of the estrous cycle?
(A) Proestrus
(B) Estrus
(C) Anestrus
(D) Diestrus

2013

192. Abortion due to infectious causes often presents with diagnostic fetal lesions. Equine Herpesvirus-1 (EHV-1) abortion typically features which of the following pathognomonic gross lesions in the aborted fetus?
(A) Severe hydrocephalus with marked arthrogryposis
(B) Leather-like thickened skin with diffuse alopecia
(C) Pinpoint hepatic necrosis and lung edema
(D) Massive multifocal hemorrhage in the fetal brain

2024

193. Orchitis in bulls is a severe condition. Brucella abortus infection in bulls typically causes which specific pathological sequence in the male reproductive tract?
(A) Primary severe prostatitis followed by acute urethritis
(B) Necrotizing epididymitis and orchitis
(C) Primary neoplastic transformation of interstitial Leydig cells
(D) Chronic fibrous stricture of the preputial orifice

2019

194. Basic cellular responses to neural injury include astrogliosis. Which specific intermediate filament protein is heavily expressed and used as an immunohistochemical marker for reactive astrocytes?
(A) Vimentin filament
(B) Desmin protein
(C) Cytokeratin marker
(D) GFAP

2009

195. Spongiform encephalopathies are universally fatal. Which specific method is absolutely required to definitively confirm a diagnosis of Rabies virus (a different encephalitic condition) in animal brain tissue post-mortem?
(A) DFA testing for viral antigen
(B) Gross pathologic examination of brain for hemorrhage
(C) Aerobic bacterial culture of cerebrospinal fluid
(D) Standard complete blood count analysis

2021

196. Osteochondrosis is characterized by a failure of endochondral ossification. When a piece of necrotic articular cartilage and underlying subchondral bone breaks free into the joint space, it is termed:
(A) An osteophyte
(B) An enthesophyte
(C) A joint mouse
(D) A pannus

2019

197. Exertional rhabdomyolysis in horses is characterized by massive muscle damage. Which intrinsic muscle protein is released into the blood and subsequently filtered by the kidneys, posing a high risk for toxic acute tubular necrosis?
(A) Myoglobin
(B) Globular actin
(C) Heavy chain myosin
(D) Cardiac troponin

2022

198. Goiter pathogenesis is deeply linked to iodine. Besides dietary iodine deficiency, the ingestion of which class of compounds found in plants of the Brassica family can induce goiter by interfering with iodine uptake?
(A) Pyrrolizidine alkaloids
(B) Cardiac glycosides
(C) Oxalates
(D) Goitrogens

2006

199. Diabetes mellitus pathology in dogs differs fundamentally from that in cats. The vast majority of canine diabetes cases are akin to human Type 1 diabetes. What is the fundamental pathological basis for this?

Click Here to Buy book on Flipkart

37. (A) Pars esophagea
Gastric ulcers in pigs are highly prevalent and occur almost exclusively in the pars esophagea, a non-glandular region of the stomach. Unlike the glandular mucosa, this stratified squamous epithelium lacks a protective mucus-bicarbonate barrier. Consequently, ingestion of finely ground feed or stressful environments increases acid splashing, leading to rapid epithelial erosion, ulceration, and potential hemorrhage.

38. (D) Aflatoxicosis
While centrilobular necrosis is the most common pattern of toxic hepatic injury, midzonal necrosis is a unique and less common pattern. This specific distribution of hepatocellular damage is classically observed in livestock, particularly horses and swine, suffering from acute or subacute aflatoxicosis. It results from the regional distribution of metabolic enzymes capable of biotransforming aflatoxin.

39. (B) Pancreatic acinar atrophy
Exocrine pancreatic insufficiency in dogs, particularly German Shepherds, is most commonly caused by pancreatic acinar atrophy. This condition is an immune-mediated disease characterized by selective T-lymphocyte-directed destruction of the pancreatic acinar cells. While the endocrine islets remain intact, the loss of enzyme-producing acinar tissue leads to severe maldigestion, steatorrhea, and weight loss.

40. (C) Fibrosis, mineralization, and functional parenchymal loss
An end-stage kidney represents the common terminal pathway of diverse chronic renal diseases. Macroscopically, these kidneys are shrunken, pale, and firm. Histologically, the original diagnostic features are obliterated by a triad of diffuse interstitial fibrosis, widespread dystrophic mineralization of tubules, and severe functional parenchymal loss, making it impossible to determine the primary initiating cause.

41. (D) Deposition of circulating immune complexes in the glomerular capillary walls
Glomerulonephritis in domestic animals is predominantly a type III hypersensitivity reaction. This process is initiated by the chronic deposition of circulating antigen-antibody complexes within the glomerular capillary basement membrane. Once deposited, these immune complexes activate the complement cascade, which recruits and activates neutrophils. This secondary inflammatory response damages the filtration barrier, leading to proteinuria and glomerulosclerosis.

42. (A) Struvite
Struvite uroliths, composed of magnesium ammonium phosphate, form preferentially in alkaline urine. In dogs, this condition is strongly associated with lower urinary tract infections caused by urease-producing bacteria like Staphylococcus pseudintermedius. The bacterial urease enzyme hydrolyzes urea into ammonia, which increases the urine pH and provides the ammonium ions required for struvite precipitation.

43. (B) Erythritol
Brucella abortus exhibits a strong tissue tropism for the pregnant uterus and placenta in ruminants. This localization is mediated by erythritol, a four-carbon sugar alcohol produced in high concentrations by the bovine placenta and fetal fluids. Erythritol acts as a powerful growth stimulant for the bacterium, leading to massive bacterial replication, necrotizing placentitis, and subsequent abortion.

44. (B) Escherichia coli
Escherichia coli is the leading coliform pathogen responsible for environmental mastitis in dairy cows. The severe systemic and local signs of this infection are driven by the release of endotoxin from the cell wall of dying bacteria. This lipopolysaccharide triggers a massive inflammatory cascade, leading to localized vasculitis, thrombosis, hemorrhagic necrotizing tissue damage, and endotoxemic shock.

45. (A) Both A and R are true and R is the correct explanation of A.
Wallerian degeneration is the stereotypical response of a peripheral or central nerve fiber to transection. When an axon is severed, the distal segment is completely disconnected from the neuronal cell body. This separation halts the axoplasmic transport of essential structural proteins and organelles, causing the distal axon and its myelin sheath to disintegrate and undergo phagocytosis.

46. (C) Microglia
Neuronophagia is a pathognomonic microscopic marker of active, irreversible neuronal injury

NODIA

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

leukocytes and alveolar walls in this disease is mediated by which specific virulence factor?
(A) Alpha toxin
(B) Enterotoxin
(C) Leukotoxin
(D) Shiga-like toxin

2005

209. Pulmonary edema visually presents as heavy, wet, and congested lungs. In cases of acute, severe alveolar edema, a characteristic macroscopic finding in the trachea and major bronchi upon necropsy is:
(A) Dry, hyperemic mucosa with no exudate
(B) Stable frothy fluid
(C) Thick, green, caseous pus blocking the airways
(D) Solid, laminated fibrinous casts

2007

210. In the pathology of the oral cavity, ruminal acidosis has systemic consequences. The systemic absorption of lactic acid and endotoxins from the damaged ruminal mucosa frequently leads to a severe secondary vascular pathology in the hooves of cattle known as:
(A) Contagious foot rot
(B) Interdigital hyperplasia
(C) Pedal osteitis
(D) Laminitis

NODIA

****

ANSWERS

1. (B) Ventricular septal defect
Ventricular septal defects represent a common congenital cardiac anomaly where an opening exists in the interventricular septum. This structural defect allows oxygenated blood to flow from the left ventricle directly into the right ventricle, causing a left-to-right shunt. Over time, the increased pulmonary blood volume leads to arterial remodeling, pulmonary hypertension, and shunt reversal.

2. (D) Fibrinous pericarditis
Traumatic reticulopericarditis in cattle occurs when a sharp foreign object penetrates the reticulum, diaphragm, and pericardium. This mechanical trauma introduces opportunistic bacteria into the pericardial space, triggering a severe inflammatory response. The hallmark of this condition is a massive accumulation of fibrin, leading to the classic bread-and-butter appearance of fibrinous pericarditis.

3. (A) Endothelial injury
In strongyle-induced cranial mesenteric arteritis, larval migration of Strongylus vulgaris causes severe damage to the arterial wall. According to Virchow's triad, endothelial injury is the most critical initiator of thrombosis in this localized pathology. The disruption of the endothelial barrier exposes subendothelial collagen, which triggers platelet adhesion, activation, and subsequent local thrombus formation.

4. (C) Classical Swine Fever
Multiple marginal splenic infarcts are highly characteristic and diagnostically significant lesions in pigs infected with Classical Swine Fever virus. The virus targets endothelial cells, leading to vasculitis, thrombosis, and subsequent ischemic necrosis. Because the splenic circulation is terminal, these vascular occlusions result in distinct, wedge-shaped, dark red infarctions along the edges of the spleen.

5. (C) A is true but R is false.
Thymomas are primary neoplasms of the thymic epithelial cells rather than the lymphoid components. While these tumors are frequently associated with secondary paraneoplastic syndromes like myasthenia gravis and exfoliative dermatitis, the autoantibodies are not directly produced by the tumor cells. Instead, the neoplastic epithelial cells fail to properly educate T-lymphocytes, leading to autoimmune dysfunction.

6. (D) Loss of glycosaminoglycans and hypocellularity in tracheal rings
Tracheal collapse in toy breed dogs is primarily a degenerative condition characterized by the progressive loss of structural integrity in the hyaline cartilage rings. Affected rings exhibit a significant decrease in glycosaminoglycan and chondroitin sulfate content, alongside chondrocyte hypocellularity. This biochemical alteration reduces the cartilage's water-binding capacity, making the tracheal rings soft and prone to dorsoventral flattening.

7. (B) Fibrinous bronchopneumonia
Fibrinous bronchopneumonia is characterized by cranioventral consolidation of the lungs, extensive fibrin deposition on the pleural surface, and lobular hepatization. In ruminants, infections with Mannheimia haemolytica typically produce this lesion, marked histologically by necrotic leukocytes known as oat cells. These specialized cells result from the cytotoxic effects of the bacterial leukotoxin on alveolar macrophages.

8. (C) Diffuse alveolar damage with hyaline membrane formation
In Acute Respiratory Distress Syndrome, the critical structural lesion impairing gas exchange is diffuse alveolar damage. This pathology is initiated by severe endothelial and alveolar epithelial injury, leading to increased vascular permeability. Consequently, protein-rich fluid escapes into the alveoli and condenses with necrotic cellular debris, forming thick, pink hyaline membranes that severely block oxygen diffusion.

9. (D) Lactic acid
Ruminal acidosis is initiated by the sudden ingestion of highly fermentable carbohydrates, which alters the microbial ecosystem of the rumen. This dietary shift promotes the rapid

Downloaded from www.pdf.tube

proliferation of Streptococcus bovis and Lactobacillus species. These bacteria ferment carbohydrates to produce excessive amounts of lactic acid, drastically lowering the ruminal pH and causing chemical injury to the mucosal lining.

10. (A) Macrophages
The characteristic thickened, corrugated intestinal mucosa of Johne's disease is caused by chronic granulomatous enteritis. This persistent inflammation is driven by the intracellular survival of Mycobacterium avium subspecies paratuberculosis. The organism replicates within the lamina propria and submucosa, recruiting massive numbers of epithelioid macrophages and multinucleated giant cells, which physically distort and expand the mucosal folds.

11. (B) Severe hypoxia or passive venous congestion due to right-sided heart failure
Centrilobular hepatocytes are highly susceptible to hypoxic injury because they are located at the distal end of the hepatic sinusoidal perfusion pathway, receiving the lowest oxygen concentration. Consequently, systemic hypoxia, severe anemia, or passive venous congestion resulting from right-sided heart failure leads to selective degeneration and necrosis of these centrilobular cells.

12. (B) Trypsinogen
Acute hemorrhagic necrotic pancreatitis is initiated by the abnormal, premature activation of trypsinogen to trypsin within the pancreatic acinar cells. Once trypsin is activated, it triggers a destructive cascade by activating other digestive zymogens, including elastase and phospholipases. This enzymatic cascade leads to autodigestion of the pancreatic parenchyma, severe vascular damage, hemorrhage, and localized fat necrosis.

13. (A) Tubulorrhexis and multifocal tubular damage
Ischemic acute tubular necrosis is characterized by tubulorrhexis, which is the disruption of the tubular basement membrane. This mechanical compromise occurs patchily along various segments of the nephron, unlike toxic injury which typically leaves the basement membrane intact. The destruction of this structural scaffold severely hinders the organized regeneration of the damaged tubular epithelial cells.

14. (C) Epsilon toxin of Clostridium perfringens type D
Pulpy kidney disease in sheep is caused by the systemic absorption of epsilon toxin produced by Clostridium perfringens type D. This potent angiotoxin targets endothelial cells, increasing vascular permeability throughout the body. In the kidneys, this causes rapid postmortem autolysis of the renal cortex, while in the brain, it leads to bilateral symmetrical encephalomalacia.

NODIA

15. (A) P-3, Q-4, R-1, S-2
Feline lower urinary tract disease is commonly associated with idiopathic sterile inflammation and matrix plugs. Emphysematous cystitis arises from gas-producing bacterial infections utilizing glucose in diabetic patients. Enzootic hematuria in cattle is directly linked to chronic ingestion of toxic bracken fern. Struvite urolithiasis in dogs frequently develops secondary to urease-producing bacterial urinary tract infections.

16. (C) Progesterone-mediated cystic endometrial hyperplasia
The development of canine pyometra is fundamentally linked to the luteal phase of the estrous cycle, during which progesterone levels remain elevated. Progesterone stimulates endometrial gland secretion, reduces uterine contractions, and suppresses local uterine immunity. This hormonal influence leads to cystic endometrial hyperplasia, creating an ideal, fluid-rich environment for secondary bacterial colonization, most commonly by Escherichia coli.

17. (B) Dihydrotestosterone
Benign prostatic hyperplasia in aging, intact male dogs is driven by hormonal imbalances, specifically an altered estrogen-to-testosterone ratio. Within the prostate, the enzyme 5-alpha-reductase converts circulating testosterone into dihydrotestosterone, a highly active androgen. Dihydrotestosterone binds to nuclear receptors on prostatic cells, stimulating local growth factors that induce diffuse stromal and epithelial hyperplasia.

18. (D) Swollen cell body, peripheral nucleus, and dispersed Nissl substance
Central chromatolysis is a highly characteristic sublethal response of neurons to axonal injury or metabolic stress. Microscopically, the cell body swells due to water influx, and the nucleus is displaced toward the periphery. Additionally,

retinal damage and a characteristic cupping of the optic nerve head.

56. (C) Intercellular edema within the epidermis
Spongiosis is a common histopathological feature of acute and subacute inflammatory skin diseases. It is characterized by the accumulation of extracellular fluid within the intercellular spaces of the epidermis. This fluid accumulation pushes the keratinocytes apart, making the desmosomal attachments highly visible under the microscope, resembling a sponge-like network within the spinous layer.

57. (D) Persistent shunt between pulmonary artery and descending aorta
During fetal life, the ductus arteriosus diverts blood from the pulmonary artery directly into the aorta, bypassing the non-functional lungs. A patent ductus arteriosus occurs when this vascular shunt fails to close shortly after birth. This persistent communication allows oxygenated blood from the high-pressure aorta to flow back into the low-pressure pulmonary artery.

58. (A) Interstitial myocarditis with intranuclear inclusions
Neonatal puppies infected with canine parvovirus-2 can develop a severe, acute myocarditis if infected during the first few weeks of life when myocardial cells are rapidly dividing. The virus replicates within the active cardiomyocytes, causing cell death. Histologically, this presents as interstitial myocarditis characterized by basophilic intranuclear inclusion bodies within surviving myocardial cells.

59. (B) Strongylus vulgaris
In horses, verminous arteritis and aneurysms are caused by the migration of Strongylus vulgaris larvae. The larvae ingest vascular endothelium and migrate into the intima of the cranial mesenteric artery. This presence causes severe endothelial damage, intimal hyperplasia, and thrombosis, which weakens the arterial wall and leads to focal dilations or aneurysms.

60. (B) Umbilical vein
Omphalophlebitis, commonly referred to as navel ill, is an inflammation of the umbilical vein in neonatal farm animals. It occurs when environmental bacteria contaminate the unhealed umbilical cord shortly after birth. This infection can quickly track along the umbilical vein to the liver, causing hepatic abscesses, or enter the systemic circulation, leading to septicemia.

61. (A) Diffuse granulomatous or lymphoid hyperplasia
Splenomegaly can be classified as either congestive or firm. A firm, meaty consistency on the cut surface indicates cellular proliferation rather than blood pooling. This firm splenomegaly is typically driven by diffuse granulomatous inflammation, neoplastic infiltration, or marked lymphoid hyperplasia. These processes expand the splenic parenchyma with cells, making the organ resistant to collapsing.

62. (C) T-cell lymphoma
Feline leukemia virus is a retrovirus that infects hematopoietic cells in cats. The virus integrates into the host genome, leading to insertional mutagenesis and oncogene activation. This genetic disruption primarily drives the malignant transformation of lymphoid cells, most commonly resulting in the development of T-cell lymphomas, particularly the thymic or mediastinal forms in young cats.

63. (D) Maxillary tooth root disease
In horses, the roots of the caudal maxillary cheek teeth project directly into the maxillary sinuses. Consequently, apical tooth infections or periodontal disease can easily breach the thin alveolar bone separating the teeth from the sinus cavity. This anatomical relationship makes maxillary tooth root disease the most common cause of secondary unilateral sinusitis in equine patients.

64. (C) Alveolar septa
Interstitial pneumonia is characterized by inflammation that primarily targets the alveolar septa rather than the alveolar spaces. It is usually caused by blood-borne pathogens or inhaled toxic gases. Histologically, the alveolar walls become thickened by edema, inflammatory cell infiltration, and subsequent fibrosis, which severely compromises gas exchange across the blood-air barrier.

65. (A) I and III
Pulmonary edema involves the accumulation of fluid within the alveoli, compromising respiration. Heart failure cells are alveolar macrophages containing phagocytosed hemosiderin, indicating chronic pulmonary

Click Here to Buy book on Amazon

for lesions on the tricuspid valve of the right side of the heart. This localization frequently leads to septic pulmonary embolism and subsequent right-sided heart failure.

28. (B) Canine
Atherosclerosis is exceptionally rare in most domestic animals due to differences in lipid metabolism. However, dogs affected by chronic, severe hypothyroidism or diabetes mellitus can develop significant atherosclerotic lesions. The lack of thyroid hormones impairs lipid clearance, causing marked hypercholesterolemia. This results in the accumulation of lipids and foam cells within the media and intima of systemic arteries.

29. (A) Corynebacterium pseudotuberculosis
Caseous lymphadenitis in sheep and goats is caused by Corynebacterium pseudotuberculosis, a facultative intracellular pathogen. The bacterium induces a chronic granulomatous necrotizing lymphadenitis. The characteristic onion-ring appearance of the lesions is produced by successive waves of necrosis followed by the deposition of concentric layers of fibrous connective tissue as the host attempts to wall off the infection.

30. (C) Vasodilation → Margination → Diapedesis → Chemotaxis → Phagocytosis
The acute inflammatory response begins with rapid local vasodilation, which slows blood flow and allows leukocytes to move to the vessel periphery, a process called margination. Leukocytes then transmigrate through the endothelial wall via diapedesis. Following chemical gradients through chemotaxis, these cells migrate to the site of injury, where they finally perform phagocytosis of pathogens.

31. (D) Bracken fern
Ingestion of bracken fern in cattle causes a variety of toxic syndromes depending on the duration of exposure. Chronic consumption of the plant's active principle, ptaquiloside, directly damages the rapidly dividing cells of the bone marrow. This toxic insult leads to severe myelosuppression, resulting in pancytopenia, clinical aplastic anemia, and life-threatening hemorrhagic diathesis.

NODIA

32. (C) Pasteurella multocida
Atrophic rhinitis in swine is a synergistic disease involving Bordetella bronchiseptica and toxigenic strains of Pasteurella multocida. The dermonecrotic toxin produced by Pasteurella multocida is the primary driver of osteolytic damage. This toxin inhibits osteoblast differentiation and stimulates osteoclast activity, leading to progressive resorption and atrophy of the nasal turbinates and facial deformity.

33. (B) Blastomyces dermatitidis
Blastomyces dermatitidis is a dimorphic fungus that causes systemic mycosis in dogs, primarily targeting the lungs. The yeast phase of the organism in tissues is characterized by thick, refractile double-contoured cell walls and broad-based budding. This fungal pathogen elicits a strong pyogranulomatous inflammatory response, characterized by clusters of neutrophils surrounded by macrophages and giant cells.

34. (A) Centriacinar emphysema
In horses with recurrent airway obstruction, chronic airway inflammation and bronchoconstriction lead to incomplete exhalation. This air trapping increases pressure within the distal airways. The resulting alveolar emphysema is typically centriacinar, where the destructive changes are localized to the respiratory bronchioles and alveolar ducts, leaving the distal alveolar sacs relatively intact until advanced stages.

35. (B) II and IV
Primary ruminal tympany is classically associated with the ingestion of high-protein legumes, which forms a stable foam, rather than grain. Furthermore, Fusobacterium necrophorum is a bacterium involved in necrobacillosis and calf diphtheria, not a primary viral pathogen. In contrast, vesicular stomatitis clinically mimics foot-and-mouth disease, and secondary tympany involves physical obstruction of eructation.

36. (C) Early postpartum weeks
Left displaced abomasum occurs most frequently during the early postpartum weeks in high-producing dairy cows. The transition from a pregnant state to lactation is marked by a sudden increase in abdominal space, abomasal hypomotility due to high-grain diets, and metabolic imbalances. These combined factors allow the abomasum to shift anatomically from the ventral floor to the left.

(A) Severe peripheral insulin resistance with fully intact beta cells
(B) Absolute insulin deficiency due to beta-cell destruction
(C) Islet amyloidosis leading to delayed insulin secretion and release
(D) Hypersecretion of glucagon by active pancreatic alpha cell tumors

2003

200. Keratitis refers to inflammation of the cornea. A specific, chronic, progressive, immune-mediated superficial keratitis common in German Shepherd dogs that begins at the lateral limbus and spreads centrally is known as:
(A) Descemetocele
(B) Feline eosinophilic keratitis
(C) Pannus (chronic superficial keratitis)
(D) Acute infectious bovine keratoconjunctivitis

2022

201. Which of the following dermatological lesions is described as a small, circumscribed, solid elevation of the skin (less than 1 cm in diameter)?
(A) Macule
(B) Vesicle
(C) Plaque
(D) Papule

2024

202. The Tetralogy of Fallot is a complex congenital anomaly. The primary developmental defect that initiates the four characteristic features of this condition is which of the following?
(A) Asymmetrical conotruncal septation
(B) Failure of closure of the ductus arteriosus
(C) Complete agenesis of the tricuspid valve
(D) Premature closure of the foramen ovale

2009

203. Traumatic reticulopericarditis is initiated by a foreign body. Which anatomical feature of the bovine stomach predisposes the animal to this specific condition?
(A) Intense muscular contractions of the rumen pushing objects cranially
(B) The honeycomb structure of the reticulum
(C) Lack of a protective stratified squamous epithelium in forestomach
(D) The extremely thin muscularis layer of the distal abomasum

2010

204. Atherosclerosis is histologically characterized by the accumulation of lipid, inflammatory cells, and fibrous tissue in the arterial wall. In which specific layer of the artery do the pathognomonic "atheromatous plaques" primarily develop?
(A) Tunica adventitia
(B) Tunica intima
(C) Tunica media
(D) Vasa vasorum

2015

205. Consider the following statements regarding lymph node and spleen pathologies:
I. Splenomegaly with a bloody consistency is often due to congestion from barbiturate euthanasia.
II. Lymphadenitis typically results in small, fibrotic, and pale lymph nodes during the acute phase.
III. Anthrax causes severe splenic engorgement and a characteristic "blackberry jam" appearance.
IV. Splenic infarcts are invariably fatal within 24 hours.
V. Lymphomas in dogs are most frequently of the multicentric form.
Which of the above statements are INCORRECT?
(A) II and IV
(B) I, III and V
(C) II, IV and V
(D) I and II

2019

206. Bone marrow pathology includes leukemias. A critical histological distinction between a lymphocytic leukemia and a multicentric lymphoma with secondary blood involvement is primarily based on which factor?
(A) The size of the lymphocytes
(B) The presence of mitotic figures
(C) The primary site of origin
(D) The response to corticosteroid therapy

2012

207. Tracheitis is commonly infectious. In dogs, "kennel cough" is a highly contagious tracheobronchitis. Which of the following bacteria is a primary recognized etiologic agent in this complex?
(A) Streptococcus equi subspecies
(B) Pasteurella multocida isolates
(C) Mycobacterium bovis strain
(D) Bordetella bronchiseptica

2025

208. Fibrinous bronchopneumonia is a severe, often fatal disease in cattle, classically caused by Mannheimia haemolytica. The severe necrosis of

Downloaded from www.pdf.tube

in the central nervous system. When a neuron undergoes necrosis, it releases chemotactic signals that recruit local microglia. These resident macrophages of the brain migrate to the damaged cell, cluster around it, and physically engulf the necrotic debris, effectively clearing the damaged neuron.

47. (D) Pyramidal cells of the hippocampus
Negri bodies are diagnostic, round-to-oval, eosinophilic intracytoplasmic inclusion bodies found in rabies-infected animals. These structures consist of viral nucleocapsid proteins accumulating within the cytoplasm of infected neurons. While they can be found in various regions, they are most consistently and abundantly identified within the pyramidal cells of the hippocampus in carnivores.

48. (C) Rickets involves failure of mineralization of both the physis and osteoid in growing animals, whereas osteomalacia involves failure of osteoid mineralization in adults with closed physes.
Rickets and osteomalacia are metabolic bone diseases caused by vitamin D or phosphorus deficiency. The key difference lies in the skeletal maturity of the affected animal. Rickets occurs in growing animals, affecting both the cartilage of the physis and newly formed osteoid. In contrast, osteomalacia occurs in adults with closed growth plates, solely affecting osteoid remodeling.

49. (B) Elbow (humeroradial) joint
Osteochondrosis is a focal disturbance of endochondral ossification that occurs in rapidly growing animals. In swine, the disease is highly prevalent and has a strong breed predisposition. While multiple joints can be subclinically involved, the medial humeral condyle of the elbow joint is the most common anatomical site for clinical osteochondrosis dissecans lesions.

50. (D) Metaplastic bone formation within injured skeletal muscle
Myositis ossificans is a rare, non-neoplastic condition characterized by the abnormal development of bone within skeletal muscle. This process is initiated by localized muscle trauma or chronic irritation. The resulting tissue damage triggers a metaplastic response, wherein local fibroblasts and mesenchymal stem cells differentiate into osteoblasts, depositing osteoid and mineralized bone in the muscular parenchyma.

51. (A) Zenker's degeneration and necrosis of myofibers
Exertional rhabdomyolysis in horses is characterized by acute muscle damage triggered by exercise. Histologically, the classic lesion is Zenker's degeneration, where the skeletal muscle fibers lose their cross-striations, swell, and undergo coagulative necrosis. This myofiber destruction is accompanied by the release of myoglobin into the blood, leading to pigmentary nephropathy and dark-colored urine.

52. (C) Low calcium, high phosphorus
Nutritional secondary hyperparathyroidism is caused by diets with a low calcium content or an excessively high phosphorus-to-calcium ratio, such as all-meat or high-grain diets. This mineral imbalance leads to persistent hypocalcemia. The parathyroid glands respond to the low calcium levels by undergoing hyperplasia and secreting excessive parathyroid hormone, driving systemic bone resorption.

53. (A) Pituitary Pars Intermedia Dysfunction
Adenomas of the pituitary pars intermedia are common in older horses and represent the underlying pathology of Pituitary Pars Intermedia Dysfunction. The loss of hypothalamic dopaminergic inhibition allows the melanotropes of the pars intermedia to proliferate and overproduce pro-opiomelanocortin-derived peptides. This hormonal excess leads to systemic signs including hirsutism, laminitis, muscle wasting, and polyuria.

NODIA

54. (D) Immune-mediated cortical atrophy
Hypoadrenocorticism in dogs is predominantly caused by bilateral, immune-mediated destruction of the adrenal cortex. This autoimmune process leads to progressive, severe atrophy of all three cortical zones, sparing the adrenal medulla. The loss of mineralocorticoid and glucocorticoid secretion results in the classic clinical signs of aldosterone and cortisol deficiency, such as electrolyte imbalances.

55. (A) P-3, Q-4, R-1, S-2
Cataracts are characterized by a loss of lens transparency, disrupting light transmission. Keratitis involves corneal inflammation, often accompanied by vascularization. Otitis media denotes inflammation of the tympanic bulla in the middle ear. Glaucoma is defined by elevated intraocular pressure, which causes progressive

the rough endoplasmic reticulum disassembles, resulting in the dispersion and apparent loss of the basophilic staining Nissl substance from the center.

19. (A) Perivascular mononuclear cuffing
Transmissible spongiform encephalopathies are degenerative diseases characterized by neuronal vacuolation, reactive astrogliosis, and the accumulation of abnormal prion proteins. Because these diseases are non-inflammatory, they do not elicit a classical inflammatory response in the brain parenchyma. Consequently, perivascular mononuclear cuffing, which is typical of viral encephalitides, is completely absent in spongiform encephalopathies.

20. (C) Secondary hyperparathyroidism
Osteodystrophia fibrosa is a metabolic bone disorder caused by sustained elevation of parathyroid hormone, typically arising from nutritional or renal secondary hyperparathyroidism. This excess hormone stimulates osteoclast activity, leading to generalized bone resorption. To compensate for the lost bone mineral, the body deposits immature fibrous connective tissue, resulting in weakened, enlarged, and deformed bones.

21. (A) Sarcoplasmic reticulum
In nutritional myopathy, a deficiency in selenium or vitamin E impairs the cellular antioxidant defense system. This deficiency permits uncontrolled free-radical damage to membrane lipids, particularly targeting the sarcoplasmic reticulum and mitochondrial membranes. The structural disruption of the sarcoplasmic reticulum causes a massive influx of extracellular calcium into the sarcoplasm, triggering myofibrillar hypercontraction and necrosis.

22. (D) Follicular cell hypertrophy and hyperplasia
Primary goiter due to iodine deficiency is initiated by a lack of thyroid hormone synthesis, which fails to exert negative feedback on the pituitary gland. In response, the pituitary releases excessive amounts of thyroid-stimulating hormone. This hormonal stimulation drives the follicular cells of the thyroid gland to undergo marked hypertrophy and hyperplasia, resulting in diffuse glandular enlargement.

23. (B) Islet amyloidosis
The key morphological hallmark of feline diabetes mellitus is islet amyloidosis, characterized by the extracellular deposition of amylin within the pancreatic islets of Langerhans. Amylin is co-secreted with insulin by beta cells; however, chronic overproduction and misfolding lead to its accumulation as amyloid fibrils. This deposition physically disrupts and progressively destroys the insulin-producing beta cells.

24. (C) Retinal ganglion cell loss and optic disc cupping
In chronic glaucoma, a persistent elevation in intraocular pressure impairs perfusion to the delicate inner structures of the eye. This sustained mechanical and ischemic stress causes progressive degeneration and loss of the retinal ganglion cells. Simultaneously, the elevated pressure pushes the optic nerve head backward, leading to a characteristic excavation known as optic disc cupping.

25. (A) I and II
Hyperkeratosis represents an abnormal thickening of the stratum corneum due to increased keratin production or delayed desquamation. Parakeratosis is characterized by the retention of nuclei within these corneal cells, signaling abnormal epidermal maturation. Conversely, acanthosis is defined as hyperplasia of the stratum spinosum, whereas intercellular edema within the epidermis is referred to as spongiosis.

26. (A) Activation of the renin-angiotensin-aldosterone system
In chronic congestive heart failure, decreased cardiac output leads to reduced renal perfusion. The kidneys respond by activating the renin-angiotensin-aldosterone system, which promotes renal sodium and water retention. This physiological mechanism aims to expand blood volume to maintain blood pressure; however, the persistent volume overload increases hydrostatic pressure, ultimately driving fluid extravasation and systemic edema.

27. (B) Tricuspid valve
In cattle, bacterial endocarditis is most commonly caused by Trueperella pyogenes or Staphylococcal species. Unlike other domestic species where the mitral valve is primarily affected, cattle exhibit a unique predisposition

Click Here to Buy book on Flipkart

congestion and microhemorrhages. Conversely, atelectasis refers to the incomplete expansion or collapse of alveoli, not their abnormal distension with air, which is instead the defining feature of pulmonary emphysema.

66. (D) Accumulation of fluid or air in the pleural cavity
Compression atelectasis occurs when the lung parenchyma is physically compressed by space-occupying lesions or fluid accumulation outside the lungs. The presence of pleural effusion, pyothorax, or air within the pleural cavity increases intrapleural pressure, overcoming the natural distending pressure of the lungs. This external force collapses the alveoli, preventing them from expanding during inspiration.

67. (A) Mucosal breach from chemical ruminitis
The ruminitis-liver abscess complex begins with ruminal acidosis, which lowers the ruminal pH and damages the stratified squamous epithelium. This chemical injury causes mucosal erosion and ulceration. Opportunistic bacteria, primarily Fusobacterium necrophorum, colonize these damaged sites, breach the mucosal barrier, enter the portal circulation, and travel to the liver, where they establish pyogenic abscesses.

68. (C) Mucosal congestion and superficial errors
Aside from anatomical displacement to the left, a displaced abomasum exhibits specific pathological changes on gross inspection. The malpositioning compromises venous drainage while arterial supply remains intact, leading to passive venous congestion of the abomasal wall. This vascular compromise results in mucosal edema, hemorrhage, and superficial mucosal erosions or ulcers due to local ischemia.

69. (A) Crypt epithelial cells
Lawsonia intracellularis is the causative agent of proliferative enteropathy in swine. This obligate intracellular bacterium specifically infects the cytoplasm of actively dividing crypt epithelial cells. The infection prevents these cells from maturing as they migrate up the villi, driving uncontrolled crypt cell hyperplasia. This pathological process leads to marked thickening of the mucosal wall.

70. (B) II, III, I, IV
The pathogenesis of hepatic cirrhosis follows a strict chronological sequence of events. Chronic, irreversible hepatocyte necrosis triggers a persistent inflammatory response. This injury stimulates hepatic stellate cells to undergo myofibroblastic transformation, depositing diffuse fibrous tissue. Surviving hepatocytes proliferate to form regenerative nodules, which disrupt hepatic architecture, compress vasculature, and ultimately cause portal hypertension.

71. (B) It receives the highest toxin concentration from incoming blood
Periportal hepatocytes, located in zone one of the hepatic acinus, are closest to the incoming blood supply from the portal vein and hepatic artery. Consequently, they are exposed to the highest concentrations of oxygen, nutrients, and absorbed toxins. Toxins that do not require metabolic activation to cause damage will selectively destroy these periportal cells first.

72. (C) Pancreas and duodenum
Feline triaditis is a well-recognized clinical syndrome characterized by concurrent inflammation of three closely associated abdominal organs. Because the pancreatic duct and the common bile duct in cats share a single opening into the duodenum, ascending bacterial infections can easily spread. This anatomical arrangement links cholangiohepatitis with pancreatitis and inflammatory bowel disease in the duodenum.

73. (D) Firm, nodular, shrunken, and fibrotic
Chronic pancreatitis is characterized by progressive, irreversible destruction of the pancreatic parenchyma. As the active inflammatory response subsides, the necrotic acinar tissue is replaced by dense fibrous connective tissue. Macroscopically, this leads to a pancreas that is firm, nodular, shrunken, and fibrotic, with minimal functional exocrine tissue remaining to produce digestive enzymes.

74. (A) Tubular obstruction by cellular casts
The reduction of glomerular filtration rate in acute tubular necrosis is driven by several synergistic mechanisms. Sloughed, necrotic tubular epithelial cells accumulate within the lumen, forming dense cellular casts that physically obstruct urine outflow. This obstruction increases intratubular pressure, which directly opposes the hydrostatic filtration pressure in the glomerulus, severely reducing overall filtration.

Downloaded from www.pdf.tube

75. (D) IV only
Interstitial nephritis primarily targets the renal tubules and the surrounding interstitial connective tissue, rather than the glomeruli. Leptospirosis in dogs is a classic example of this pathology. White spotted kidneys in calves represent focal areas of chronic interstitial nephritis. Because the primary insult spares the glomeruli initially, labeling the glomerulus as the primary target is incorrect.

76. (B) The vesicoureteral valve mechanism
Ascending pyelonephritis occurs when bacteria migrate from the urinary bladder up to the renal pelvis. The primary anatomical barrier preventing this retrograde flow of urine is the vesicoureteral valve mechanism. When this valve is compromised due to inflammation, urinary tract obstruction, or congenital defects, vesicoureteral reflux occurs, allowing bacteria to ascend into the kidneys.

77. (A) Struvite crystals and proteinaceous matrix
Urethral obstruction in male cats with lower urinary tract disease is most commonly caused by urethral plugs. These plugs are semi-solid materials composed of microscopic struvite crystals embedded within a thick, sticky proteinaceous matrix. This organic matrix consists of desquamated epithelial cells, inflammatory proteins, and mucus, which easily occlude the narrow feline urethra.

78. (C) Small, raised, gray-white mucosal lymphoid nodules
Follicular cystitis is a chronic form of urinary bladder inflammation. It is characterized grossly by the presence of multiple, small, raised, gray-white mucosal nodules. These nodules represent hyperplastic lymphoid follicles within the lamina propria, which develop in response to persistent antigenic stimulation from chronic, low-grade bacterial infections of the lower urinary tract.

79. (D) Luteinizing Hormone (LH)
Cystic ovarian disease in dairy cattle typically manifests as large, fluid-filled follicular cysts. These structures develop when a mature follicle fails to rupture and ovulate. This failure is fundamentally caused by a lack of the preovulatory luteinizing hormone surge from the pituitary gland, which is necessary to trigger ovulation and subsequent luteinization of the follicle.

NODIA

80. (C) Periglandular fibrosis
In mares, chronic endometritis and endometrosis are significant causes of infertility. The hallmark microscopic change of this chronic condition is periglandular fibrosis, where concentric layers of fibrous tissue surround the endometrial glands. This fibrosis physically restricts glandular secretion, preventing the production of histotroph, which is essential for supporting the early developing embryo.

81. (B) Dark red, necrotic cotyledons with characteristic white calcified foci
Abortion in sheep caused by Toxoplasma gondii produces highly characteristic placental lesions. On gross examination, the intercotyledonary membranes appear normal, but the cotyledons are dark red, swollen, and contain numerous small, distinct, white calcified foci of necrosis. These multifocal necrotic lesions represent sites of active protozoal replication and subsequent tissue destruction.

82. (D) Suppurative to granulomatous mastitis
Chronic mastitis caused by Staphylococcus aureus is characterized by a suppurative to granulomatous inflammatory response. The bacteria survive within the mammary tissue, inducing localized necrosis and abscess formation. Over time, the host attempts to wall off these lesions with fibrous tissue, resulting in hard, fibrotic quarters containing microabscesses and a permanent loss of milk production.

83. (A) Brucella ovis
Brucella ovis is the primary infectious agent responsible for contagious epididymitis in rams. The bacterium exhibits a specific tropism for the male reproductive tract, localizing in the epididymis. This infection induces a chronic, progressive, suppurative to granulomatous epididymitis, which frequently causes unilateral or bilateral testicular atrophy, poor semen quality, and permanent infertility.

84. (C) Tubular necrosis and subsequent interstitial fibrosis
Severe purulent orchitis is characterized by extensive neutrophilic infiltration and tissue destruction within the testis. This acute inflammatory response causes widespread tubular necrosis, destroying the delicate seminiferous epithelium. As the lesion heals, the damaged tubules are replaced by extensive

interstitial fibrosis, which permanently halts spermatogenesis and leads to irreversible sterility.

85. (C) A is true but R is false.
Astrogliosis is a classic chronic response to central nervous system injury, leading to the formation of a glial scar. While astrocytes undergo hypertrophy and hyperplasia to wall off the damaged area, they do not deposit collagen to bridge the defect. Collagen deposition is restricted to the meninges and perivascular spaces; glial scars consist of intermediate filaments.

86. (D) Mesencephalic aqueduct (Aqueduct of Sylvius)
Non-communicating hydrocephalus is characterized by the accumulation of cerebrospinal fluid due to a physical obstruction within the ventricular system. The narrowest point in this pathway is the mesencephalic aqueduct. Consequently, congenital stenosis, cellular debris, or inflammatory exudate at this specific site easily blocks fluid flow, leading to dilation of the lateral and third ventricles.

87. (B) Brilliant autofluorescence
Polioencephalomalacia in ruminants leads to cerebrocortical necrosis, typically associated with thiamine deficiency or sulfur toxicity. When fresh brain slices from an affected animal are exposed to ultraviolet light, the necrotic cortical tissue exhibits a brilliant autofluorescence. This diagnostic phenomenon is caused by the accumulation of lipid breakdown products and lipofuscin within the damaged tissue.

88. (C) Osteoclasts
Osteopetrosis is a rare, congenital bone disease characterized by a generalized increase in bone mass. This pathology results from a functional defect in osteoclasts, which fail to resorb and remodel bone during endochondral ossification. Consequently, the primary spongiosa persists and fills the medullary cavity, producing dense, heavy bones that are paradoxically brittle and prone to fracture.

89. (D) Hematogenous spread from omphalophlebitis or enteritis
Suppurative arthritis in neonatal farm animals, commonly known as joint ill, is most frequently a sequela of hematogenous bacterial spread. Pathogens enter the neonatal circulation via contaminated umbilicus or damaged intestinal mucosa. These bacteria lodge in the highly vascular synovial membrane and epiphyseal capillaries, triggering an acute inflammatory response characterized by purulent joint effusion.

90. (A) Affected muscle shows discrete pale, chalky-white streaks or patches.
Nutritional myopathy is characterized by distinct macroscopic changes in skeletal and cardiac muscle. Affected muscle fibers undergo coagulative necrosis and dystrophic mineralization, giving them a dry, chalky appearance. Macroscopically, this presents as discrete pale, chalky-white streaks or patches that run parallel to the myofibers, contrasting sharply with the uniform dark red color of normal muscle.

91. (B) Functional adenoma of a single parathyroid gland
Primary hyperparathyroidism in dogs is an uncommon endocrine disorder characterized by autonomous, unregulated secretion of parathyroid hormone. This condition is most frequently caused by a benign, functional adenoma involving a single parathyroid gland. The excess hormone stimulates osteoclast activity and renal calcium reabsorption, leading to persistent hypercalcemia, hypophosphatemia, and generalized osteocytic osteolysis.

92. (B) Symmetrical bilateral alopecia and epidermal atrophy
Hyperadrenocorticism in dogs causes widespread dermatological lesions due to the catabolic effects of excess cortisol. Cortisol inhibits collagen synthesis and follicular activity, leading to progressive follicular atrophy. This endocrine pathology presents clinically as symmetrical, bilateral alopecia, epidermal atrophy, and fragile skin. Additionally, dystrophic mineralization of dermal collagen, known as calcinosis cutis, may develop.

93. (A) Adrenal medulla
A pheochromocytoma is a neuroendocrine neoplasm that originates from the chromaffin cells of the adrenal medulla. These cells are embryologically derived from the neural crest and are responsible for synthesizing and secreting catecholamines, such as epinephrine and norepinephrine. Neoplastic proliferation of

Click Here to Buy book on Amazon

152. (C) ALT is specific for hepatocyte injury in dogs and cats.
In dogs and cats, alanine aminotransferase is a highly specific cytoplasmic enzyme found in high concentrations within hepatocytes. Because of this localization, any damage that alters hepatocyte cell membrane permeability leads to rapid leakage of the enzyme into the bloodstream. This makes it an invaluable serum marker for detecting active hepatocellular injury in veterinary clinical diagnostics.

153. (D) Hepatic stellate cell
During chronic hepatic injury, inflammatory cytokines stimulate the activation of quiescent hepatic stellate cells residing in the perisinusoidal space of Disse. Once activated, these cells transdifferentiate into contractile, myofibroblast-like cells. They synthesize and deposit excessive amounts of extracellular matrix proteins, primarily type I and III collagen, leading to progressive hepatic fibrosis.

154. (A) Serum TLI
The trypsin-like immunoreactivity test is the gold standard diagnostic assay for exocrine pancreatic insufficiency in dogs. This test measures the concentration of trypsinogen and trypsin circulating in the bloodstream. Since trypsinogen is produced exclusively by the pancreas, a severely low serum concentration reflects a profound loss of pancreatic acinar tissue, confirming the diagnosis.

155. (B) Imperfect regeneration with interstitial fibrosis
The tubular basement membrane serves as the essential scaffold for orderly epithelial cell replacement after acute renal tubular injury. When this basement membrane is completely destroyed, a process termed tubulorrhexis, the structural guide for organized cellular alignment is lost. Consequently, the epithelial regeneration becomes highly disorganized, resulting in persistent interstitial fibrosis.

156. (B) C5a
In the pathogenesis of glomerulonephritis, the activation of the complement cascade by immune complexes yields several biologically active fragments. Among these, C5a is a highly potent anaphylatoxin and chemotactic factor. It binds to receptors on circulating neutrophils, actively recruiting them into the glomerular capillaries, where they release destructive lysosomal enzymes and reactive oxygen species.

157. (A) Neurogenic inflammation related to stress
Feline idiopathic cystitis is a complex, multi-systemic disorder where environmental stress plays a central role. Stress triggers excessive sympathetic nervous system activation, which stimulates sensory neurons in the bladder wall. This neurogenic inflammation causes the release of neuropeptides, such as substance P, inducing mast cell degranulation, mucosal vasodilation, and severe submucosal edema.

158. (C) Corpus luteum
In cows, pyometra typically develops following postpartum contamination of the uterus when the animal enters a luteal phase. The persistence of a functional corpus luteum on the ovary produces progesterone, which closes the cervix and reduces uterine immune defenses. This allows the trapped bacterial infection to persist, leading to the accumulation of purulent exudate.

159. (D) Inflammation of all layers of the uterine wall
Metritis is defined as acute inflammation involving all anatomical layers of the uterine wall, including the endometrium, submucosa, muscularis, and serosal layers. This severe disease condition typically occurs postpartum following dystocia, retained placenta, or bacterial contamination. It is characterized by severe systemic illness, toxemia, and a foul-smelling uterine discharge in affected cattle.

160. (C) Elevated somatic cell count
Subclinical mastitis is a highly prevalent form of mammary gland inflammation that lacks any visible abnormalities in the milk or udder. It is primarily detected by monitoring the somatic cell count in milk samples. An elevated somatic cell count, composed mainly of neutrophils, indicates an active immune response to an intramammary pathogen.

161. (B) Prostatic cyst formation and abscessation
Benign prostatic hyperplasia is a common age-related condition in intact male dogs driven by altered androgen-to-estrogen ratios. The hyperplastic glandular tissue can develop microcysts that subsequently coalesce into large intraprostatic cysts. These fluid-filled structures are prone to secondary bacterial infection,

NODIA

typical initial response of the upper respiratory tract to mild viral infections or physical irritants. The nasal discharge is clear to cloudy, lacking significant numbers of neutrophils.

104. (C) Left recurrent laryngeal nerve
Laryngeal hemiplegia in horses is a neurogenic muscle atrophy caused by progressive axonopathy of the left recurrent laryngeal nerve. Due to its exceptionally long anatomical course around the aortic arch, this nerve is highly susceptible to mechanical stretching and compression. Its degeneration leads to atrophy of the cricoarytenoideus dorsalis muscle, preventing vocal fold abduction.

105. (A) I and III
Bronchopneumonia typically originates at the transition between the bronchioles and alveoli, where inhaled pathogens settle. Fibrinous pneumonia is characterized by severe vascular injury, allowing fibrinogen to leak into the parenchyma. In contrast, interstitial pneumonia is usually caused by hematogenous pathogens or toxic injury to the alveolar wall, not typically by inhaled bacteria.

106. (A) Gastric acid irritation and mixed bacterial infection
Aspiration of acidic stomach contents combined with oral bacteria creates a highly destructive pulmonary environment. The gastric acid causes direct chemical burns on the delicate alveolar and bronchial linings, facilitating bacterial invasion. This synergistic action leads to rapid tissue death, gangrenous necrosis, and putrefactive decomposition of the affected lung parenchyma.

107. (B) Increased alveolar capillary permeability
Non-cardiogenic pulmonary edema arises from direct damage to the blood-air barrier rather than hydrostatic pressure changes. When the permeability of the alveolar and capillary membranes increases, fluid, proteins, and inflammatory cells leak directly into the alveoli. This compromise of structural integrity prevents efficient gas exchange and triggers severe respiratory dysfunction in affected patients.

108. (B) "Bloat line" along the esophageal mucosa
Severe ruminal distension exerts massive pressure on the diaphragm, which compresses the thoracic organs and vasculature. This compression severely restricts venous return, leading to congestion in the head and neck while the thoracic esophagus remains pale. The resulting demarcated boundary of congestion and pallor along the esophageal mucosa is a reliable diagnostic indicator.

109. (A) Abomasal volvulus leading to ischemia and necrosis
Right displaced abomasum constitutes a critical surgical emergency because it frequently rotates along its mesenteric axis. This rotational movement results in a complete abomasal volvulus, which immediately compromises local venous drainage and arterial blood supply. The resulting acute ischemia rapidly causes hemorrhagic infarction, transmural necrosis, and potential rupture of the gastric wall.

110. (A) Grain ingestion → Proliferation of Streptococcus bovis → Fall in ruminal pH → Proliferation of Lactobacilli → Lactic acidosis
Rapid ingestion of highly fermentable carbohydrates alters the ruminal microflora, promoting the proliferation of amylolytic Streptococcus bovis. This microbial shift rapidly lowers the ruminal pH, creating an acidic environment that inhibits normal flora while encouraging the growth of acid-tolerant Lactobacilli. The subsequent accumulation of lactic acid leads to severe systemic metabolic acidosis.

111. (D) Johne's Disease
Mycobacterium avium subspecies paratuberculosis infects the intestinal mucosa of ruminants, triggering a persistent, cell-mediated immune response. This chronic infection results in granulomatous enteritis, characterized by massive infiltration of macrophages and giant cells into the lamina propria. The consequence is a severely thickened, corrugated intestinal wall that prevents proper nutrient absorption.

112. (C) Negative energy balance during the periparturient period
During the early postpartum period, dairy cows undergo a severe negative energy balance because energy demands for milk production outpace dietary intake. This triggers massive mobilization of non-esterified fatty acids from adipose tissue to the liver. The hepatocytes convert these fatty acids into triglycerides, which accumulate inside the liver cells, causing fatty degeneration.

Downloaded from www.pdf.tube

these cells leads to uncontrolled hormone release, causing systemic hypertension and tachycardia.

94. (C) Melting ulcer (keratomalacia)
A melting ulcer is a deep, rapidly progressive corneal ulcer characterized by the enzymatic digestion of the corneal stroma. This destructive process is driven by collagenases and proteases released from invading bacteria, particularly Pseudomonas, or from local inflammatory cells. The enzymatic breakdown liquefies the corneal stroma, giving it a characteristic gelatinous, melting appearance.

95. (A) P-3, Q-4, R-1, S-2
Acanthosis refers to the increased thickness of the stratum spinosum due to hyperplasia. Hyperkeratosis is defined as the thickening of the stratum corneum. Parakeratosis is characterized by the retention of nuclei within the stratum corneum cells. Exocytosis describes the active migration of inflammatory cells, such as neutrophils, into the epidermis.

96. (C) Loss of cohesion between epidermal cells
Acantholysis is the primary pathological mechanism in autoimmune skin diseases like pemphigus. This process involves the loss of cohesion between epidermal keratinocytes due to the immunological destruction of desmosomes by autoantibodies. The disrupted cellular connections cause the keratinocytes to detach and round up, leading to intraepidermal clefting, vesicle formation, and superficial ulceration.

97. (B) Pulmonary hypertension and edema
Left-sided congestive heart failure impairs the heart's ability to pump blood from the pulmonary circulation into the systemic system. This dysfunction causes blood to back up into the left atrium and pulmonary veins, increasing hydrostatic pressure. The resulting pulmonary venous congestion and hypertension force fluid into the pulmonary interstitium and alveoli, causing pulmonary edema.

98. (D) Primary pulmonary or pulmonary vascular disease
Cor pulmonale is defined as hypertrophy or dilation of the right ventricle resulting from diseases that affect the lungs or pulmonary vasculature. Conditions such as chronic alveolar emphysema, dirofilariasis, or pulmonary fibrosis increase resistance to blood flow through the lungs. This sustained pulmonary hypertension forces the right ventricle to work harder, leading to compensatory hypertrophy.

NODIA

99. (A) Fibrinous
Uremic pericarditis is a classic extrarenal lesion observed in animals with severe, acute, or end-stage chronic renal failure. The accumulation of systemic uremic toxins damages the vascular endothelium of the pericardial capillaries, increasing permeability. This permits the leakage of high-molecular-weight fibrinogen, which polymerizes into fibrin, giving the epicardial surface a rough, shaggy, fibrinous appearance.

100. (B) Tunica media
Equine Viral Arteritis is caused by an arterivirus that exhibits a strong tropism for endothelial cells and smooth muscle cells of small arteries. The virus replicates within these cells, causing segmental necrotizing arteritis. This inflammatory process primarily targets the tunica media, leading to necrosis of smooth muscle, increased vascular permeability, localized edema, and petechial hemorrhages.

101. (A) Mediastinal form
The mediastinal form of lymphoma is a common presentation in cats, often strongly associated with feline leukemia virus infection. This neoplasm arises in the thymus or mediastinal lymph nodes, presenting as a large space-occupying thoracic mass. The growing tumor compresses the thoracic vasculature and lymphatic ducts, frequently causing pleural effusion or chylothorax.

102. (D) Massive proliferation of non-lymphoid precursors in marrow and blood
Myeloid leukemia is a neoplastic disorder originating from non-lymphoid hematopoietic stem cells within the bone marrow. The hallmark of this condition is the uncontrolled, massive proliferation of granulocytic, erythroid, monocytic, or megakaryocytic precursors. These neoplastic cells crowd out normal marrow elements, leading to cytopenias, and frequently infiltrate peripheral blood and solid organs.

103. (B) Mild viral infections
Catarrhal rhinitis is an acute, superficial inflammation of the nasal mucosa characterized by the hypersecretion of mucus from goblet cells and mucosal glands. This pathology represents the

thick-walled structures continuously secrete high levels of progesterone, which exerts negative feedback on the hypothalamus, preventing estrus and causing persistent anestrus.

123. (B) Escherichia coli
The canine pyometra complex is initiated by prolonged progesterone stimulation during diestrus, which induces cystic endometrial hyperplasia and suppresses uterine immunity. This uterine environment allows opportunistic bacteria to colonize and proliferate rapidly. Escherichia coli is the most common isolate, as its virulent strains possess specific adhesins that allow binding to progesterone-sensitized endometrium.

124. (B) Tritrichomonas foetus
Tritrichomonas foetus is a venereal protozoan pathogen in cattle that colonizes the reproductive tract after mating. It causes early embryonic death or fetal mortality, often without immediate expulsion of the conceptus. The retained fetus undergoes maceration within the uterine lumen under anaerobic conditions, culminating in post-coital pyometra and severe reproductive failure in herds.

125. (C) A is true but R is false.
Coliform mastitis produces severe systemic shock because of the massive release of lipopolysaccharide endotoxins into the blood from the mammary gland, rather than direct necrosis of the secretory epithelium. While the local tissue damage is relatively mild, the systemic absorption of endotoxins triggers a profound inflammatory response, vascular pooling, hypotension, and toxemia.

126. (C) Fibrous adhesions of the vaginal tunics
Chronic epididymitis triggers a persistent, severe inflammatory response that extends beyond the epididymis to involve the surrounding tunics. This active inflammation leads to fibrin deposition and subsequent organization, resulting in dense fibrous adhesions between the parietal and visceral vaginal tunics. These adhesions restrict testicular mobility and severely impair critical heat regulation mechanisms.

127. (D) Gitter cells
Gitter cells represent the fully activated, phagocytic state of microglia within the central nervous system. Following focal parenchymal necrosis, such as encephalomalacia, these cells migrate to the injured area and engulf myelin breakdown products and lipid debris. Their cytoplasm becomes highly vacuolated and distended with lipids, giving them a characteristic foamy appearance.

128. (C) Neurons of the brainstem obex
Bovine Spongiform Encephalopathy is a progressive neurodegenerative disease caused by prions. The characteristic spongiform change, consisting of clear, round vacuoles within the neuronal perikaryon and axonal processes, has a highly consistent anatomical distribution. These lesions are predominantly located in the brainstem, with the obex being the key site for post-mortem surveillance.

129. (B) Nodular 'rachitic rosary' enlargements
Rickets results from a deficiency of vitamin D or phosphorus, leading to a failure of provisional calcification of cartilage during endochondral ossification. The persistent cartilage at the growth plates fails to mature, resulting in a widened and disorganized chondrocyte zone. At the costochondral junctions, this excessive cartilaginous growth produces visible nodular enlargements called a rachitic rosary.

130. (D) Fibrinous-proliferative arthritis
Erysipelothrix rhusiopathiae in swine is a classical cause of chronic polyarthritis, characterized by persistent joint pathology. The pathogen triggers a severe, long-term immune response within the synovial membrane, leading to prominent hyperplasia of synovial villi and deposition of fibrin. This fibrinous-proliferative arthritis causes progressive joint deformity, periarticular fibrosis, and restricted mobility.

131. (A) They express a unique Type 2M myosin isoform.
Masticatory muscle myositis is a canine autoimmune disease targeting the temporalis, masseter, and pterygoid muscles. These muscles are embryologically and structurally unique because they express a specific type 2M myosin isoform not found in skeletal limbs. Autoantibodies specifically attack this 2M fiber type, triggering severe eosinophilic infiltration, inflammation, and eventual myofiber atrophy.

Click Here to Buy book on Flipkart

113. (B) Portal hypertension and acquired portosystemic shunts
Chronic hepatic injury leads to diffuse bridging fibrosis and regenerative nodule formation, which severely distorts the normal vascular architecture of the liver. This structural disruption increases resistance to blood flow through the hepatic portal system, resulting in portal hypertension. Consequently, the body develops acquired portosystemic shunts to bypass the congested liver.

114. (D) Chronic fibrosing cholangitis
Fasciola hepatica parasites reside within the bile ducts of ruminants, causing physical trauma and releasing immunogenic secretions. This chronic irritation induces a severe inflammatory and proliferative response in the duct walls, characterized by extensive connective tissue deposition. The resulting chronic fibrosing cholangitis leads to marked thickening, mineralization, and stenosis of the biliary system.

115. (A) I, II and III
Acute pancreatitis is characterized by the premature activation of digestive enzymes, which leads to widespread enzymatic fat necrosis in the peripancreatic tissue. Over time, repeated bouts of inflammation and tissue destruction can progress to chronic pancreatitis. This chronic damage causes irreversible loss of acinar cells, culminating in exocrine pancreatic insufficiency, maldigestion, and steatorrhea.

116. (C) Nephron
The nephron functions as the primary anatomical and physiological unit of the kidney. Because the glomerulus and its corresponding renal tubule share an intimate structural and vascular relationship, primary damage to one component invariably compromises the survival and function of the other. Consequently, chronic glomerulonephritis eventually leads to tubular atrophy and interstitial fibrosis.

117. (A) Ethylene glycol
Ingestion of ethylene glycol leads to rapid hepatic metabolism into highly toxic compounds, primarily oxalic acid. Oxalic acid binds to systemic calcium, forming insoluble calcium oxalate crystals that precipitate within the lumens of the proximal convoluted tubules. This crystal deposition causes physical obstruction and direct cytopathic damage, resulting in acute tubular necrosis.

NODIA

118. (D) Membranous glomerulonephritis
Membranous glomerulonephritis is characterized by the diffuse thickening of the glomerular capillary basement membrane, caused by the subepithelial deposition of immune complexes. Unlike other proliferative types, there is a notable absence of inflammatory or mesangial cell infiltration. This structural compromise alters the filtration barrier, resulting in severe proteinuria and nephrotic syndrome.

119. (B) Purulent exudate in the pelvis radiating into the parenchyma
Acute pyelonephritis typically arises from an ascending bacterial infection originating in the lower urinary tract. The bacteria colonize the renal pelvis, inducing a severe suppurative inflammatory response. Macroscopically, this is visible as accumulation of purulent exudate within the pelvis, which then radiates outward into the medulla and cortex along the renal tubules.

120. (C) Supersaturation of urine with the crystalloid
The development of uroliths is a complex chemical process where the fundamental and mandatory prerequisite is the supersaturation of urine with specific mineral crystalloids. When the concentration of these dissolved minerals exceeds their solubility limit, they begin to precipitate out of solution. This precipitation initiates crystal nucleation, growth, and subsequent aggregation into uroliths.

121. (D) Cystitis cystica
Chronic irritation and inflammation of the urinary bladder mucosa can stimulate the nesting and downward proliferation of epithelial cells. These epithelial nests, known as von Brunn's nests, subsequently undergo cystic degeneration, forming small, fluid-filled cystic cavities. This specific morphological response is termed cystitis cystica and is visible microscopically as multiple raised mucosal nodules.

122. (A) Luteal cysts
In swine, ovarian cysts are classified based on their histological characteristics and hormone production. Luteal cysts develop when unruptured follicles undergo partial luteinization of the granulosa and theca cell layers. These

142. (C) Copper
Copper is an essential cofactor for the enzyme lysyl oxidase, which mediates the cross-linking of collagen and elastin fibers in arterial walls. In pigs, a dietary deficiency of copper compromises this cross-linking, causing arterial fragility. The weakened aorta is highly susceptible to structural failure, leading to dissecting aneurysms and fatal internal hemorrhage.

143. (D) Lymphadenitis – Inflammation of the lymph node
Lymphadenitis represents the specific inflammatory response within a lymph node, typically triggered by infectious pathogens such as bacteria, viruses, or fungi. It must be differentiated from simple reactive hyperplasia or neoplastic processes like lymphoma. The prefix "lymphaden-" refers specifically to the lymph node, while the suffix "-itis" denotes active inflammation.

144. (C) B and T lymphocytes
Severe combined immunodeficiency in Arabian foals is an autosomal recessive disorder caused by a mutation in the DNA-dependent protein kinase gene. This genetic defect prevents the normal rearrangement of antigen receptor genes during development. Consequently, the foal is completely unable to produce mature B and T lymphocytes, leaving it highly vulnerable to fatal infections.

145. (C) II and IV
Sinusitis is routinely accompanied by rhinitis due to the direct anatomical communication between the nasal passages and the sinuses. Furthermore, structural airway obstructions such as laryngeal paralysis primarily compromise inspiration rather than expiration. This is because the negative airway pressure generated during inhalation causes collapse of the unsupported, paralyzed laryngeal cartilages.

146. (D) Bronchioloalveolar junction
Inhaled bacterial pathogens primarily settle at the bronchioloalveolar junction, which is the most vulnerable zone in the respiratory tract. At this site, the protective mucociliary apparatus ends, and the airflow velocity drops abruptly, allowing bacteria to deposit. Additionally, the local defense mechanisms must transition from mechanical clearance to alveolar macrophage phagocytosis.

NODIA

147. (A) Hypostatic atelectasis
Hypostatic atelectasis is a form of lung collapse commonly seen in recumbent, bedridden, or anesthetized animals. Prolonged recumbency causes constant gravitational pooling of blood and fluids in the dependent lung portions. This congestion compresses the small airways and alveoli, while reducing local ventilation, leading to progressive alveolar collapse in the lower lung fields.

148. (C) Spongiosis and cell ballooning in the stratum spinosum
Vesicular stomatitis is initiated by viral replication within the stratified squamous epithelial cells of the oral cavity. This intracellular infection causes cell swelling and fluid accumulation, known as ballooning degeneration and spongiosis. As these damaged cells rupture and fluid accumulates between remaining cells, intraepithelial vesicles and bullae form within the stratum spinosum.

149. (A) Soluble leaf proteins
Primary ruminal tympany, or frothy bloat, is driven by the rapid digestion of young, succulent legumes. These plants contain high concentrations of soluble leaf proteins, which act as foaming agents in the rumen. The proteins create a stable, viscous foam that traps fermentation gases, preventing normal eructation and causing life-threatening ruminal distension.

150. (A) III, I, II, IV
The pathogenesis of left displaced abomasum follows a chronological sequence. It begins with abomasal hypomotility or atony, which prevents normal emptying and leads to the accumulation of fermentation gases. This gas accumulation increases buoyancy, causing the abomasum to displace to the left side, resulting in a partial obstruction of abomasal outflow.

151. (B) Cyclooxygenase
Non-steroidal anti-inflammatory drugs cause gastric ulceration primarily through the systemic and local inhibition of the cyclooxygenase enzymes. These enzymes are crucial for synthesizing cytoprotective prostaglandins, which maintain mucosal blood flow, stimulate bicarbonate and mucus secretion, and limit acid production. Their absence compromises the mucosal barrier, leading to direct acid-induced tissue necrosis.

132. (C) Lymphocytic thyroiditis
Primary hypothyroidism in dogs is most frequently caused by lymphocytic thyroiditis, an immune-mediated condition characterized by infiltration of lymphocytes, plasma cells, and macrophages into the thyroid parenchyma. This inflammatory process triggers progressive, irreversible destruction of thyroid follicles and replacement of the glandular tissue with fibrous connective tissue, leading to hormone deficiency.

133. (A) Decreased calcitriol synthesis
Chronic renal failure impairs the kidney's ability to synthesize active calcitriol because of the loss of functioning renal parenchyma and enzyme activity. This lack of calcitriol reduces calcium absorption in the gastrointestinal tract, causing systemic hypocalcemia. In response, the parathyroid glands undergo marked hyperplasia and increase parathyroid hormone secretion to restore blood calcium levels.

134. (D) Functional pituitary adenoma secreting ACTH
Naturally occurring canine hyperadrenocorticism is overwhelmingly pituitary-dependent, caused by a functional ACTH-secreting pituitary adenoma in the pars distalis or pars intermedia. The excessive, unregulated secretion of ACTH stimulates the adrenal cortex, causing bilateral hyperplasia of the zona fasciculata and zona reticularis. This hyperplasia results in persistent, systemic overproduction of cortisol.

135. (A) P-3, Q-1, R-4, S-2
Endocrine pathologies present with highly characteristic features. Diabetes mellitus is defined by clinical signs of polyuria, polydipsia, and glucosuria. Hypoadrenocorticism results in aldosterone loss, causing hyponatremia and hyperkalemia. Hyperthyroidism in geriatric cats causes weight loss despite polyphagia, while pituitary pars intermedia dysfunction in horses is the primary cause of hirsutism.

136. (C) Malassezia pachydermatis
Malassezia pachydermatis is a lipid-dependent, non-mycelial yeast commonly residing on canine skin and within the external ear canal. Under abnormal conditions of altered microclimate or local immunity, this opportunist proliferates rapidly, causing severe erythematous otitis externa. Cytological examination reveals characteristic single-budding, oval yeasts with a distinct footprint or peanut-like physical morphology.

137. (D) Uveitis
Uveitis represents a major ocular pathology defined as inflammation of the uveal tract, which comprises the iris, ciliary body, and choroid. This condition is triggered by infectious diseases, trauma, or systemic immune-mediated processes. It leads to clinical signs such as aqueous flare, miosis, corneal edema, and can progress to permanent blindness.

138. (A) Acanthosis
Acanthosis is a specific dermatopathological term referring to an increase in the thickness of the stratum spinosum of the epidermis. This epidermal hyperplasia is commonly a protective response to chronic irritation, friction, or persistent inflammation. It is characterized by cellular proliferation within the prickle cell layer, resulting in thickened skin.

139. (B) Staphylococcus pseudintermedius
Superficial pustular dermatitis, commonly known as impetigo, is a bacterial infection of the subcorneal epidermis. In dogs, this condition is almost exclusively caused by Staphylococcus pseudintermedius, a major skin commensal and opportunist. The bacteria colonize the superficial skin layer, releasing specific chemotactic factors that recruit host neutrophils, forming fragile epidermal pustules.

140. (B) Tetralogy of Fallot
The Tetralogy of Fallot is a classic congenital cardiac defect characterized by four primary abnormalities. These include a high ventricular septal defect, dextroposition of the aorta, pulmonic stenosis, and right ventricular hypertrophy. The combination of these structural defects causes significant shunting of deoxygenated blood and leads to persistent systemic tissue hypoxia.

141. (A) Left ventricular subendocardium
Nutritional myopathy in calves and lambs, resulting from selenium or vitamin E deficiency, causes oxidative damage to active muscular tissues. While skeletal muscles are extensively involved, cardiac lesions present as discrete, chalky pale plaques of necrosis and calcification. These cardiac lesions are classically concentrated in the left ventricular subendocardium region of the heart.

Downloaded from www.pdf.tube

leading to abscessation, extreme pelvic pain, and physical compression of the rectum.

162. (D) Growth cone
Wallerian degeneration describes the process of axonal breakdown following physical injury. After axonal transection, the distal segment degenerates, while the proximal axonal stump seals and forms a specialized structure called a growth cone. This dynamic, amoeboid tip is highly enriched in cytoskeletal elements and receptors that guide regeneration toward target tissues.

163. (A) Oligodendrocytes
Canine distemper virus is a highly pantropic pathogen that can cause progressive demyelination within the central nervous system. The virus directly infects and replicates inside oligodendrocytes, the glial cells responsible for synthesizing and maintaining the myelin sheath. This cellular infection leads to metabolic disruption, cell death, and subsequent primary demyelinating lesions.

164. (C) Three-dimensional conformational structure
The pathogenic prion protein is the infectious agent responsible for transmissible spongiform encephalopathies. It shares the exact same primary amino acid sequence as the normal cellular prion protein. However, it differs fundamentally in its three-dimensional conformational structure, transitioning from a predominantly alpha-helical conformation to a highly stable, infectious beta-sheet-rich structural conformation.

165. (C) A is true but R is false.
Polioencephalomalacia is a neurodegenerative disease characterized by cortical necrosis. It can be successfully treated with thiamine if administered early, confirming the assertion. However, the reason is false because rumen microbes natively produce thiamine, whereas thiaminases are typically introduced via exogenous sources like specific weeds or produced by atypical, proliferated rumen bacteria.

166. (D) It involves the accumulation of unmineralized osteoid in adult bone.
Osteomalacia is a metabolic bone disease affecting adult animals that have closed growth plates. It is characterized by a failure in the mineralization of newly formed osteoid, which is synthesized during normal bone remodeling. This leads to the progressive accumulation of unmineralized osteoid, resulting in soft, weak bones prone to deformities.

NODIA

167. (B) Highly active skeletal muscles
Nutritional myopathy, or white muscle disease, is caused by a deficiency of selenium or vitamin E, which are critical components of cellular antioxidant systems. Highly active muscles are most severely affected because they have the highest oxygen consumption and generate the most free radicals. This active state leads to massive oxidative damage and necrosis.

168. (C) Prolonged gestation and alopecia
In neonates born to dams with severe iodine deficiency, the lack of thyroid hormones leads to profound developmental disruptions. This congenital hypothyroidism causes delayed development of the central nervous system and skeleton. Clinically, this manifests as prolonged gestation, birth of weak or dead offspring, and marked alopecia due to impaired hair follicle maturation.

169. (D) Hypercalcemia and hypophosphatemia
Primary hyperparathyroidism arises from a functional parathyroid adenoma that secretes excessive, unregulated levels of parathyroid hormone. This excess hormone directly increases osteoclastic bone resorption and renal calcium reabsorption, leading to hypercalcemia. Concurrently, it inhibits renal phosphate reabsorption in the proximal tubules, which results in significant hypophosphatemia, creating a classic diagnostic profile.

170. (A) Aldosterone
Hypoadrenocorticism, or Addison's disease, involves the bilateral destruction of the adrenal cortex, leading to a loss of aldosterone. Aldosterone normally promotes renal sodium reabsorption and potassium excretion. Its absence prevents the kidneys from conserving sodium and excreting potassium, resulting in the classic and life-threatening electrolyte imbalance of hyponatremia and severe hyperkalemia.

171. (B) Bilateral cataracts
In dogs, chronic diabetes mellitus is a common cause of bilateral cataract formation. Persistent hyperglycemia leads to high glucose levels within the aqueous humor, which diffuses into the lens. The lens enzyme aldose reductase converts

CHAPTER 10
Pathology of Infectious Diseases

2019

1. Which of the following defines bacteremia in the context of infectious disease pathology?
(A) The active multiplication of bacteria in the bloodstream leading to systemic signs
(B) The mere presence of bacteria in the bloodstream without active multiplication
(C) The circulation of bacterial toxins in the blood causing tissue damage
(D) The presence of pyogenic bacteria causing multiple abscesses in organs

2002

2. The primary portal of entry for infectious agents causing enteritis is most commonly which of the following?
(A) Respiratory tract
(B) Urogenital tract
(C) Percutaneous inoculation
(D) Gastrointestinal tract

2022

3. Septicemia is pathologically distinct from bacteremia primarily due to which characteristic?
(A) The pathogen actively multiplies within the circulatory system causing systemic illness
(B) The pathogen remains localized at the portal of entry
(C) Only bacterial toxins enter the systemic circulation
(D) The pathogen is effectively cleared by reticuloendothelial macrophages within minutes

2002

4. Toxemia frequently leads to widespread tissue damage. Which of the following lesions is most indicative of acute toxemia?
(A) Localized granulomatous inflammation
(B) Formation of caseous necrosis in the lymph nodes
(C) Diffuse petechial hemorrhages and endothelial damage
(D) Hyperkeratosis of the epidermis

2011

5. Read the given assertion and reason, and select the correct option.

Assertion (A): The respiratory tract is a common portal of entry for aerosolized viral pathogens.
Reason (R): The mucociliary escalator and alveolar macrophages fail to clear all inhaled particles, allowing some to establish infection.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2014

6. During host-pathogen interactions, which process facilitates the hematogenous dissemination of intracellular bacteria?
(A) Binding to mucosal epithelium
(B) Secretion of locally acting exotoxins
(C) Mechanical obstruction of capillaries by bacterial aggregates
(D) Phagocytosis by and survival within wandering macrophages

2003

7. The spread of an infectious agent from its initial entry site to regional lymph nodes occurs primarily through which route?
(A) Venous circulation
(B) Lymphatic drainage
(C) Arterial circulation
(D) Direct tissue invasion

2008

8. In the context of viral dissemination, what does the term 'secondary viremia' indicate?
(A) Initial shedding of viral particles into blood from primary infection sites
(B) Rapid clearance of circulating viral particles by host immune cells
(C) Systemic release of virus following replication in secondary organs
(D) Direct transmission of viral particles to adjacent epithelial layers

Click Here to Buy book on Amazon

of large, pathognomonic basophilic intranuclear inclusion bodies within hypertrophied epithelial cells.

182. (D) Gravitational settling and short, direct airways
In quadrupeds, the cranioventral lung lobes are the primary site for lesions in bacterial bronchopneumonia. This specific distribution is due to physical and physiological factors. Gravity naturally deposits inhaled particles and bacteria into these lower lobes. Furthermore, these lobes have shorter, more vertical main bronchi that facilitate direct deposition of airborne pathogens.

183. (B) 3-methylindole
Acute interstitial emphysema and edema, or fog fever, occurs in cattle moved from dry to lush pastures. These pastures contain high levels of L-tryptophan, which rumen microbes convert to 3-methylindole. Once absorbed, this compound is metabolized by pulmonary club cells into a highly reactive pneumotoxin, causing extensive necrosis of type I pneumocytes.

184. (C) A shallow, ragged erosion
Vesicular stomatitis causes intraepithelial vesicles that are highly fragile due to continuous mechanical stress in the oral cavity. These structures rapidly rupture, shedding the overlying epithelium. This leaves a shallow, ragged erosion characterized by a red, moist base. This erosion heals quickly without scarring, provided there is no secondary bacterial contamination.

185. (A) II, III and IV
Gastric ulcers in swine are localized primarily within the non-glandular pars esophagea, driven by finely ground diets. In cattle, Johne's disease causes severe granulomatous enteritis leading to a protein-losing enteropathy. In dogs, parvovirus targets the rapidly dividing crypt epithelium of the small intestine, leading to crypt necrosis and severe, hemorrhagic enteritis.

186. (A) Massive bridging necrosis
Massive bridging necrosis is a severe pattern of hepatic injury that spans adjacent vascular regions within the hepatic lobules. It typically connects central veins to other central veins or portal tracts, disrupting the hepatic architecture. This pattern occurs in late stages of acute toxic insults from hepatotoxins like pyrrolizidine alkaloids or aflatoxins.

187. (B) Steatorrhea with ravenous appetite
Exocrine pancreatic insufficiency results in a profound lack of essential digestive enzymes, such as pancreatic lipase, trypsin, and amylase. This enzymatic deficiency prevents the digestion of fats, proteins, and carbohydrates. Consequently, affected dogs present with steatorrhea, characterized by voluminous, greasy, pale stools, accompanied by a ravenous appetite and severe weight loss.

188. (B) Ulcerative glossitis and stomatitis
Uremia is the systemic clinical syndrome arising from severe renal failure, characterized by the accumulation of toxic metabolic waste products. The high circulating levels of urea are converted by oral bacteria into ammonia, causing direct chemical irritation. This ammonia production leads to the development of ulcerative glossitis and stomatitis in affected animals.

189. (A) Albumin
Glomerulonephritis compromises the selective permeability of the glomerular filtration barrier, allowing large quantities of plasma proteins to escape into the urine. Albumin is a relatively small, abundant plasma protein that is lost in the greatest clinical quantities. This severe hypoalbuminemia reduces intravascular oncotic pressure, resulting in systemic tissue edema and ascites.

190. (C) Fatal hyperkalemia
Urethral obstruction in male cats prevents the excretion of urine, causing post-renal azotemia. As urine accumulates, the kidneys are unable to excrete potassium, leading to a rapid rise in blood potassium levels. This profound hyperkalemia severely impairs myocardial electrical conduction, resulting in bradycardia, cardiac arrhythmias, and eventual fatal cardiac arrest in affected cats.

NODIA

191. (D) Diestrus
In female dogs, pyometra typically develops during diestrus, which is the phase of the estrous cycle dominated by progesterone. Progesterone stimulates endometrial gland secretion, reduces uterine contractions, and suppresses local immune function, creating an ideal environment for bacterial colonization. Opportunistic bacteria ascending from the vagina can then establish a severe uterine infection.

mechanism of ETEC (Enterotoxigenic E. coli) involves:
(A) Attaching and effacing lesion formation on mucosal microvilli
(B) Direct invasion of enterocytes leading to extensive ulceration
(C) Chronic granulomatous inflammation of the colonic mucosa
(D) Enterotoxin-mediated fluid hypersecretion

2025
42. Select the correct sequence of events in the pathogenesis of Salmonella-induced enterocolitis:
(A) Ingestion → Invasion of enterocytes and M cells → Multiplication in macrophages → Acute inflammatory response
(B) Ingestion → Multiplication in macrophages → Invasion of enterocytes → Acute inflammatory response
(C) Ingestion → Acute inflammatory response → Invasion of enterocytes → Multiplication in macrophages
(D) Invasion of enterocytes → Ingestion → Acute inflammatory response → Multiplication in macrophages

2018
43. Fibrinous bronchopneumonia with characteristic 'marbling' of the lungs in cattle is the classic gross lesion of:
(A) Pasteurella multocida strains
(B) Mannheimia haemolytica
(C) Escherichia coli bacteria
(D) Brucella abortus organisms

2016
44. The characteristic lesion of Brucellosis in the pregnant bovine uterus is:
(A) Suppurative endometritis accompanied by multiple uterine abscesses
(B) Necrotizing placentitis with leathery intercotyledonary areas
(C) Granulomatous metritis accompanied by severe chronic fibrosis
(D) Severe fibrinohemorrhagic vaginitis with mucosal erosion

2011
45. In swine, infection with Salmonella Choleraesuis typically results in:
(A) Hepatic paratyphoid nodules and colonic button ulcers
(B) Localized mild enteritis of the duodenum and jejunum
(C) Severe necrotic rhinitis accompanied by facial deformity
(D) Generalized polyserositis with no intestinal involvement

2012
46. Pasteurella multocida type D is implicated in the pathogenesis of which porcine disease characterized by turbinate atrophy?
(A) Swine Dysentery
(B) Glasser's Disease
(C) Atrophic Rhinitis
(D) Porcine Enzootic Pneumonia

2002
47. Which specific virulence factor of Escherichia coli is essential for adherence to the intestinal epithelium in neonatal calves?
(A) Endotoxin (LPS)
(B) Hemolysin toxins
(C) Shiga-like cytotoxin
(D) F5 (K99) fimbriae

2001
48. Read the following statements regarding Gram-negative infections:
I. Brucella localizes in the reticuloendothelial system.
II. Mannheimia haemolytica produces a potent leukotoxin.
III. Colisepticemia occurs commonly in calves with adequate colostral immunity.
IV. Salmonella typhimurium causes fibrino-necrotic enteritis.
V. Brucella abortus causes epididymitis in bulls.
Which of the above statements is/are incorrect?
(A) I, II and IV
(B) III
(C) III and V
(D) II, IV and V

2012
49. The presence of 'button ulcers' in the cecum and colon of pigs is highly suggestive of infection with:
(A) Erysipelothrix rhusiopathiae
(B) Salmonella serovars
(C) Pasteurella multocida
(D) Actinobacillus pleuropneumoniae

2009
50. Edema disease in pigs, caused by STEC strains of E. coli, is pathologically characterized by:
(A) Diffuse pulmonary congestion accompanied by severe alveolar edema
(B) Chronic granulomatous inflammation of the

Downloaded from www.pdf.tube

202. (A) Asymmetrical conotruncal septation
The primary developmental defect initiating the Tetralogy of Fallot is the asymmetrical division of the conotruncal septum. This embryological malformation results in unequal partition of the embryonic outflow tract, creating a narrow pulmonic trunk and a large, displaced aorta. The other three classic defects develop as a direct physiological consequence of this septal deviation.

203. (B) The honeycomb structure of the reticulum
The anatomical design of the bovine forestomach, particularly the honeycomb structure of the reticulum, predisposes cattle to traumatic reticulopericarditis. Heavy, metallic foreign bodies, such as nails or wires, tend to settle within the reticular compartment. Strong muscular contractions can then push these sharp objects through the reticular wall, migrating cranially into the pericardium.

204. (B) Tunica intima
Atherosclerosis is characterized by the formation of atheromatous plaques within the tunica intima, which is the innermost layer of the arterial wall. These plaques develop following chronic endothelial injury, leading to lipid accumulation, smooth muscle cell proliferation, and macrophage infiltration. Over time, these intimal lesions can cause vascular occlusion and severe tissue ischemia.

205. (A) II and IV
Acute lymphadenitis is characterized by enlarged, soft, and congested lymph nodes, not small, fibrotic, or pale ones, which represent chronic, inactive lesions. Additionally, splenic infarcts are typically localized, wedge-shaped lesions that are rarely fatal within twenty-four hours on their own, unless accompanied by severe, diffuse systemic diseases or massive clinical hemorrhage.

206. (C) The primary site of origin
The distinction between a primary lymphocytic leukemia and a multicentric lymphoma with secondary blood involvement is based on the primary site of origin. Leukemia originates within the hematopoietic tissues of the bone marrow, subsequently entering the peripheral blood. In contrast, lymphoma arises within solid peripheral lymphoid tissues, such as lymph nodes or the spleen.

207. (D) Bordetella bronchiseptica
Bordetella bronchiseptica is a highly contagious, gram-negative bacterium that acts as a primary etiologic agent in canine infectious respiratory disease complex. It possesses specific virulence factors, such as fimbrial adhesins and toxins, which allow it to attach to and paralyze the ciliated epithelial cells of the upper trachea, causing severe tracheobronchitis.

208. (C) Leukotoxin
Mannheimia haemolytica causes severe fibrinous bronchopneumonia in cattle, a condition mediated by its primary virulence factor, leukotoxin. This protein target-selectively binds to LFA-1 integrins on bovine leukocytes and platelets. At high concentrations, it forms transmembrane pores, causing cell lysis and releasing highly destructive lysosomal enzymes that induce severe alveolar and vascular necrosis.

209. (B) Stable frothy fluid
In cases of acute, severe pulmonary alveolar edema, fluid escapes from the capillaries and floods the alveoli. During respiration, this protein-rich fluid mixes with inspired air and surfactant, forming a stable frothy fluid. At necropsy, this froth is classically observed within the trachea and major bronchi, indicating severe ante-mortem alveolar flooding.

NODIA

210. (D) Laminitis
The systemic absorption of lactic acid and endotoxins from the damaged ruminal mucosa during acute ruminal acidosis triggers widespread vascular changes. These circulating toxins induce vasoconstriction, microvascular thrombosis, and severe inflammation within the sensitive laminae of the hooves. This pathological sequence leads to acute laminitis, causing extreme pain and severe lameness in cattle.

****

(D) IL-2 and IL-12

2012
20. Which of the following best describes the pathogenesis of enterotoxigenic tissue injury?
(A) Direct necrosis of enterocytes leading to widespread mucosal ulceration
(B) Invasion of the lamina propria leading to chronic granuloma formation
(C) Disruption of cyclic nucleotide pathways causing fluid secretion
(D) Mechanical obstruction of mucosal capillaries by bacterial emboli

2014
21. Cellular tropism of a virus is primarily determined by which of the following?
(A) Presence of specific host cell surface receptors
(B) Molecular structure and type of the viral nucleic acid
(C) Overall physical size and symmetry of the viral particle
(D) Efficiency and rate of viral genomic transcription

2002
22. Viral-induced direct cytopathic effects frequently manifest microscopically as:
(A) Extensive dermal collagen deposition
(B) Chronic granulomatous inflammation
(C) Acute neutrophilic infiltration
(D) Degeneration, swelling, and lysis

2001
23. Syncytia formation in viral infections occurs due to:
(A) Fusion of viral envelope glycoproteins with host mitochondrial membranes
(B) Fusion of adjacent cell membranes into multinucleated giant cells
(C) Rapid division of host cell nuclei in the absence of cytokinesis
(D) Phagocytosis of virus-infected cells by tissue macrophages

2010
24. Read the following statements regarding viral inclusion bodies:
I. They can be intracytoplasmic or intranuclear.
II. They represent sites of viral replication or assembly.
III. They are only seen in RNA viral infections.
IV. They are identifiable using routine light microscopy.
Which of the above statements are correct?
(A) I, II and IV
(B) I, III and IV
(C) I and II
(D) II, III and IV

2013
25. Which virus is classically known for producing distinct intracytoplasmic inclusion bodies known as Negri bodies?
(A) Canine distemper virus
(B) Poxvirus
(C) Herpesvirus
(D) Rabies virus

2001
26. Viral-induced immunosuppression is a hallmark of which of the following mechanisms?
(A) Direct lysis of target lymphoid cells
(B) Systemic hyperactivation of helper T cells
(C) Increased secretion of neutralizing antibodies
(D) Enhanced phagocytic activity of macrophages

2008
27. The formation of pathognomonic 'owl's eye' intranuclear inclusion bodies is characteristic of which viral family?
(A) Poxviridae
(B) Herpesviridae
(C) Paramyxoviridae
(D) Parvoviridae

2017
28. Which process leads to cell death in viruses exhibiting a lytic replication cycle?
(A) Integration of viral genetic material into the host genome
(B) Cell membrane rupture during virion release
(C) Persistent budding of virions without causing membrane damage
(D) Malignant transformation of the host cell into a tumor cell

2003
29. Read the given assertion and reason, and select the correct option.
Assertion (A): Poxviruses exclusively replicate in the host cell cytoplasm despite being DNA viruses.
Reason (R): Poxviruses carry their own DNA-dependent RNA polymerase, eliminating the need to enter the host nucleus.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

Click Here to Buy book on Flipkart

2001
9. Which mechanism represents a specific host adaptation to prevent pathogen entry via the gastrointestinal tract?
(A) High pH of gastric secretions
(B) Absence of lymphoid tissue in the gut
(C) Decreased peristaltic movement
(D) Low pH of gastric secretions

2016
10. The capability of a pathogen to cause disease in a susceptible host is quantitatively referred to as its:
(A) Virulence
(B) Infectivity
(C) Pathogenicity
(D) Tropism

2012
11. Which component of the Gram-negative bacterial cell wall is primarily responsible for endotoxic shock?
(A) Peptidoglycan layer
(B) Lipopolysaccharide
(C) Teichoic acid layer
(D) Outer membrane porins

2019
12. Read the following statements regarding bacterial exotoxins:
I. They are primarily secreted by living bacteria.
II. They are highly antigenic and can be converted into toxoids.
III. They are lipopolysaccharides in nature.
Which of the above statements are correct?
(A) I and II
(B) II and III
(C) I
(D) II

2013
13. Which extracellular enzyme produced by certain bacteria facilitates their spread through connective tissue by degrading hyaluronic acid?
(A) Hyaluronidase
(B) Coagulase
(C) Streptokinase
(D) Collagenase

2022
14. The bacterial capsule serves as a critical virulence factor by primarily facilitating which of the following?
(A) Enhancing bacterial motility
(B) Mediating direct attachment to host ribosomes
(C) Preventing phagocytosis by host immune cells
(D) Degrading host extracellular matrix

2013
15. Intracellular survival mechanisms of bacteria like Mycobacterium include all of the following EXCEPT:
(A) Inhibition of phagosome-lysosome fusion
(B) Resistance to lysosomal enzymes
(C) Escape from the phagosome into the cytoplasm
(D) Production of massive amounts of endotoxin

2003
16. Which bacterial mechanism specifically leads to the direct lysis of host red blood cells?
(A) Production of coagulase
(B) Release of leukocidins
(C) Secretion of hemolysins
(D) Elaboration of hyaluronidase

2009
17. Exotoxins that specifically target and damage the intestinal mucosa are classified as:
(A) Neurotoxins
(B) Enterotoxins
(C) Cytotoxins
(D) Leukotoxins

2013
18. Match the bacterial virulence factors in List I with their corresponding functions in List II.
List I: (Virulence Factor)
P. Coagulase
Q. Kinase
R. Leukocidin
S. Collagenase
List II: (Function)
1. Destroys white blood cells
2. Breaks down structural tissue proteins
3. Induces fibrin clot formation
4. Dissolves fibrin clots
(A) P-3, Q-4, R-1, S-2
(B) P-4, Q-3, R-2, S-1
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-3, S-2

2011
19. Tissue injury mediated by endotoxins typically triggers the massive release of which host cytokines?
(A) IL-1 and TNF-alpha
(B) IL-4 and IL-10
(C) IFN-beta and TGF-beta

Downloaded from www.pdf.tube

this glucose into sorbitol, an osmotically active compound that cannot cross the lens membrane, causing severe swelling.

172. (B) Extension from chronic otitis externa
Otitis media in dogs and cats most commonly occurs as a direct extension of chronic otitis externa. Persistent external ear canal inflammation can lead to the rupture of the tympanic membrane, allowing bacteria and inflammatory debris to pass directly into the tympanic cavity. This pathway initiates active suppurative inflammation of the middle ear structures.

173. (A) Stratum corneum thickening with nuclear retention
Parakeratosis is a specific dermatological alteration characterized by the abnormal maturation of epidermal cells. It is defined as the thickening of the stratum corneum with the retention of nuclei within the keratinocytes. This occurs due to accelerated epidermal turnover, preventing normal cellular differentiation and nuclear breakdown, often seen in zinc deficiency.

174. (C) Vesiculopustular dermatitis
Vesiculopustular dermatitis is a prominent morphological pattern of skin inflammation characterized by the formation of intraepidermal vesicles or pustules. This pattern is commonly observed in autoimmune diseases such as pemphigus vulgaris, where autoantibodies target desmosomal proteins. The loss of intercellular adhesion causes acantholysis, creating fluid-filled cavities within the superficial skin epidermis.

175. (A) P-3, Q-2, R-4, S-1
Gastrointestinal pathologies occur in highly specific locations within the digestive tract. Abomasal displacement is a disease affecting the glandular stomach of ruminants. Ruminal acidosis represents a pathology of the forestomach. Johne's disease predominantly targets the terminal ileum, cecum, and colon, while proliferative hemorrhagic enteropathy is localized specifically in the distal ileum.

176. (C) High membranous portion
Ventricular septal defects are common congenital cardiac anomalies in domestic animals. The interventricular septum consists of a thick muscular portion and a thin membranous portion. The high membranous portion is embryologically complex, involving the fusion of multiple endocardial cushions, making it the most frequent site of developmental failure and septal defect formation.

177. (B) Trypanosoma cruzi
In dogs, Trypanosoma cruzi, the protozoan agent of Chagas disease, has a strong tropism for myocardial tissue. The parasite invades cardiomyocytes, where it undergoes intracellular replication. This causes cell rupture and triggers a severe, diffuse, necrotizing and histiocytic myocarditis. This extensive damage often causes cardiac arrhythmias and subsequent sudden death in affected animals.

178. (D) Lines of Zahn
An arterial thrombus is formed within a dynamic, high-velocity blood flow environment. This active circulation causes the progressive deposition of platelets and fibrin, which are then covered by layers of red blood cells. On histological examination, these alternating pale and dark layers are known as the lines of Zahn, indicating antemortem formation.

NODIA

179. (A) Low-grade
Histological grading of lymphomas is based on cytological features and mitotic activity. A low-grade lymphoma is characterized by the proliferation of small, well-differentiated lymphocytes with a low mitotic index. These neoplastic cells closely resemble normal mature lymphocytes, resulting in a more slowly progressive clinical course compared to high-grade, aggressive anaplastic forms.

180. (C) Chloramphenicol
Chloramphenicol is a broad-spectrum antibiotic that can induce idiosyncratic, dose-independent bone marrow aplasia in humans, leading to fatal aplastic anemia. Because of this severe public health risk and the potential for residues to enter the food chain, its use is strictly banned or heavily restricted in food-producing animals in many countries.

181. (A) Porcine Cytomegalovirus
Inclusion body rhinitis is a common, highly contagious respiratory disease affecting young piglets. It is caused by Porcine Cytomegalovirus, a betaherpesvirus. The virus infects the epithelial cells of the nasal mucosa, leading to severe rhinitis. Histologically, it is characterized by the presence

2013
30. Which cellular event is NOT typically considered a viral-induced direct cytopathic effect?
(A) Apoptosis
(B) Lysis
(C) Amyloid deposition
(D) Syncytium formation

2006
31. The primary pathognomonic macroscopic lesion of Anthrax in cattle is:
(A) Severe fibrino-suppurative bronchopneumonia
(B) Focal hepatic necrosis with parenchymal loss
(C) Diffuse fibrinous pericarditis and effusion
(D) Splenomegaly with dark, uncoagulated blood

2003
32. Clostridium chauvoei is the causative agent of Blackleg. The characteristic microscopic lesion in affected muscle is:
(A) Diffuse mononuclear infiltration accompanied by fibrosis
(B) Granulomatous inflammation with Langhans giant cells
(C) Myonecrosis with gas bubbles and hemorrhage
(D) Segmental demyelination of peripheral nerve fibers

2011
33. Staphylococcosis in animals frequently leads to the formation of:
(A) Caseous lymphadenitis in lymph nodes
(B) Abscesses and botryomycosis
(C) Diffuse interstitial pneumonia in lungs
(D) Non-suppurative encephalitis in brain

2025
34. Listeriosis in ruminants typically presents with which primary pathological finding?
(A) Fibrinous polyserositis in body cavities
(B) Acute hemorrhagic enteritis of the small intestine
(C) Severe necrotizing lesions of skeletal muscles
(D) Microabscesses in the brainstem

2009
35. Read the following statements regarding Gram-positive bacterial pathology:
I. Anthrax bacilli produce a poly-D-glutamic acid capsule.
II. Clostridium tetani causes massive tissue necrosis and gas gangrene.
III. Listeria monocytogenes can cause late-term abortions in cattle.
Which of the above statements is/are incorrect?
(A) I and II
(B) II
(C) III
(D) I and III

2013
36. Which toxin produced by Bacillus anthracis is primarily responsible for inducing severe localized edema?
(A) Lethal factor
(B) Protective antigen
(C) Edema factor
(D) Alpha toxin

2003
37. The pathogenesis of enterotoxemia in sheep (pulpy kidney disease) is primarily due to the epsilon toxin produced by:
(A) Clostridium perfingens type D
(B) Clostridium novyi type B strain
(C) Clostridium septicum organisms
(D) Clostridium botulinum bacteria

2010
38. Microscopically, 'club-shaped' rosettes of Splendore-Hoeppli material surrounding aggregates of Gram-positive cocci in tissue sections are characteristic of:
(A) Listeriosis
(B) Actinomycosis
(C) Anthrax
(D) Botryomycosis

2021
39. Which of the following Gram-positive organisms is a classic intracellular pathogen that utilizes host actin filaments to spread from cell to cell?
(A) Staphylococcus aureus
(B) Listeria monocytogenes
(C) Bacillus anthracis
(D) Clostridium perfringens

2012
40. In cases of sudden death due to Anthrax, rigor mortis is typically:
(A) Rapidly appearing and sustained
(B) Present only in the hindlimbs
(C) Incomplete or absent
(D) Accompanied by severe muscle spasms

2012
41. Colibacillosis in young calves often manifests as white scours. The primary pathological

Click Here to Buy book on Amazon

192. (C) Pinpoint hepatic necrosis and lung edema
Equine Herpesvirus-1 is a major cause of infectious abortion in mares. The virus crosses the placenta, infecting fetal tissues and causing rapid cell death. At necropsy, the aborted equine fetus typically displays characteristic lesions, including pinpoint areas of focal necrosis within the liver, severe pulmonary edema, and prominent pleural fluid accumulation.

193. (B) Necrotizing epididymitis and orchitis
Brucella abortus is a zoonotic bacterial pathogen that has a strong tropism for the male reproductive tract of bulls. The bacteria localize within the epididymis and testes, triggering a severe, chronic inflammatory response. This leads to necrotizing epididymitis and orchitis, resulting in extensive tissue destruction, abscess formation, and permanent testicular infertility.

194. (D) GFAP
Glial fibrillary acidic protein is a class III intermediate filament protein expressed predominantly by cells of the astroglial lineage. Following injury to the central nervous system, astrocytes undergo hypertrophy and hyperplasia, a process termed astrogliosis. During this response, GFAP expression is heavily upregulated, making it a reliable immunohistochemical marker for identifying reactive astrocytes.

195. (A) DFA testing for viral antigen
To definitively confirm a diagnosis of Rabies virus post-mortem, the direct fluorescent antibody test is the gold standard method required. This assay utilizes fluorescein-labeled antibodies directed against the rabies viral nucleoprotein antigen. It is performed on fresh brain tissue smears, specifically targeting regions such as the cerebellum, hippocampus, and brainstem obex.

196. (C) A joint mouse
Osteochondrosis is a developmental orthopedic disease characterized by a focal failure of endochondral ossification in growing cartilage. In osteochondrosis dissecans, a cleft forms between the necrotic articular cartilage and underlying subchondral bone. When a piece of this cartilage and bone breaks free into the joint space, it is termed a joint mouse.

197. (A) Myoglobin
Exertional rhabdomyolysis in horses involves massive necrosis of skeletal muscle fibers, which releases large quantities of myoglobin into the circulation. The kidneys filter this abundant myoglobin, which accumulates within the renal tubules. High concentrations of myoglobin are directly nephrotoxic and cause tubular obstruction, posing a high risk for acute tubular necrosis.

198. (D) Goitrogens
Goitrogens are diverse chemical compounds found in plants of the Brassica family, such as kale and cabbage, that interfere with thyroid hormone synthesis. These substances actively inhibit the uptake of iodine by thyroid follicular cells or block the action of thyroid peroxidase. This interference prevents adequate hormone production, triggering compensatory thyroid hypertrophy.

199. (B) Absolute insulin deficiency due to beta-cell destruction
Canine diabetes mellitus is overwhelmingly caused by an absolute deficiency of insulin, resulting from the progressive destruction of pancreatic beta cells. This pathology closely resembles human Type 1 diabetes and is driven by genetic predisposition, immune-mediated pancreatitis, or progressive pancreatic acinar atrophy. Consequently, affected dogs require life-long exogenous insulin hormone replacement therapy.

NODIA

200. (C) Pannus (chronic superficial keratitis)
Pannus, or chronic superficial keratitis, is a progressive, immune-mediated corneal disease most common in German Shepherd dogs. It typically begins as a vascularized, cellular infiltrate at the lateral limbus and spreads centrally across the cornea. Chronic exposure to ultraviolet light is a key environmental trigger, causing severe, bilateral corneal tissue pigmentation.

201. (D) Papule
A papule is a fundamental dermatological lesion defined as a small, circumscribed, solid elevation of the skin that measures less than one centimeter in diameter. This lesion can arise due to localized infiltration of inflammatory cells, vascular congestion, or hyperplasia of the epidermal or dermal layers, representing an early sign of dermatitis.

meninges and brain
(C) Severe necrotizing enteritis of the small and large intestines
(D) Angiopathy causing gastric wall and mesentery edema

2007
51. The hallmark microscopic lesion of bovine tuberculosis is the:
(A) Caseating tubercle containing epithelioid and giant cells
(B) Purulent abscess surrounded by a dense fibrous connective capsule
(C) Eosinophilic granuloma containing numerous foreign body giant cells
(D) Non-caseating granuloma primarily composed of mature lymphocytes

2002
52. Johne's Disease, caused by Mycobacterium avium subsp. paratuberculosis, predominantly affects which region of the gastrointestinal tract?
(A) Stomach and duodenum
(B) Esophagus and forestomachs
(C) Ileum, cecum, and colon
(D) Rectum and anal canal

2008
53. Macroscopically, the intestinal mucosa in a severe case of Johne's Disease appears:
(A) Thickened, corrugated mucosal folds
(B) Severely ulcerated and hemorrhagic mucosa
(C) Thin, translucent, and highly friable mucosa
(D) Mucosa covered with a thick pseudomembrane

2010
54. Actinomycosis (lumpy jaw) is primarily characterized by which pathological process?
(A) Diffuse interstitial pneumonia of the pulmonary parenchyma
(B) Caseous lymphadenitis of the regional submandibular lymph nodes
(C) Acute hemorrhagic enteritis of the posterior small intestine
(D) Pyogranulomatous osteomyelitis of the jaw

2014
55. Match the acid-fast/granulomatous agents in List I with their typical lesions in List II.
List I: (Acid-Fast/Granulomatous Agent)
P. Mycobacterium bovis
Q. Mycobacterium paratuberculosis
R. Actinomyces bovis
S. Actinobacillus lignieresii
List II: (Typical Lesion)
1. Wooden tongue (granulomatous glossitis)
2. Lumpy jaw (osteomyelitis)
3. Corrugated intestinal mucosa
4. Caseating tubercles in lungs
(A) P-4, Q-3, R-2, S-1
(B) P-3, Q-4, R-1, S-2
(C) P-4, Q-2, R-3, S-1
(D) P-1, Q-3, R-2, S-4

2010
56. The 'sulfur granules' observed in the pus of Actinomycosis infections represent:
(A) Aggregates containing pure crystals of elemental sulfur
(B) Calcified necrotic remnants of degenerated parasitic ova
(C) Bacterial colonies with Splendore-Hoeppli material
(D) Debris from caseous necrosis of degenerating macrophages

2023
57. Which lipid component of the Mycobacterial cell wall is largely responsible for inhibiting phagosome-lysosome fusion and inducing granuloma formation?
(A) Lipopolysaccharide
(B) Peptidoglycan
(C) Teichoic acid
(D) Cord factor

2005
58. The epithelioid cells seen in the granulomatous lesions of tuberculosis are derived from which cell lineage?
(A) Macrophages
(B) Fibroblasts
(C) Lymphocytes
(D) Neutrophils

2010
59. In Johne's Disease, the extensive infiltration of macrophages in the lamina propria leads to primarily which clinical manifestation?
(A) Severe coughing and dyspnea
(B) Diarrhea and wasting
(C) Neurological deficits
(D) Acute systemic shock

2015
60. The characteristic microscopic lesion of Poxvirus infections in the epidermis is:
(A) Severe hyperkeratosis accompanied by minimal cellular degeneration
(B) Ballooning degeneration with intracytoplasmic inclusions
(C) Coagulative necrosis restricted solely to the

Downloaded from www.pdf.tube

2014
82. Arrange the sequence of viral spread in the pathogenesis of Rabies:
I. Replication in local muscle tissue
II. Entry via bite wound
III. Ascent via peripheral nerves to the CNS
IV. Centrifugal spread to salivary glands
Which of the following represents the correct sequence?
(A) II, I, III, IV
(B) II, III, I, IV
(C) I, II, IV, III
(D) III, II, I, IV

2024
83. The respiratory lesions in Newcastle Disease (velogenic strain) typically manifest as:
(A) Hemorrhagic tracheitis and pneumonia
(B) Chronic granulomatous pneumonia
(C) Suppurative bronchopneumonia with large abscesses
(D) Diffuse pulmonary fibrosis

2004
84. Which microscopic finding in the brain is highly characteristic of Canine Distemper encephalitis?
(A) Purulent exudate in the ventricles
(B) Symmetrical bilateral malacia of the basal ganglia
(C) Demyelination of white matter tracts
(D) Neuronal vacuolation without inflammation

2018
85. Newcastle Disease in poultry is caused by a Paramyxovirus. The viscerotropic velogenic pathotype produces pathognomonic lesions including:
(A) Hemorrhages in the proventriculus and gut lymphoid patches
(B) Massive bilateral enlargement of the sciatic nerve branches
(C) White caseous nodular lesions in the lungs and air sacs
(D) Diffuse fibrinous perihepatitis and pericarditis in birds

2001
86. In Rabies, which specific neuronal populations are most frequently evaluated for the presence of Negri bodies in carnivores and herbivores, respectively?
(A) Purkinje cells (carnivores) and Hippocampus (herbivores)
(B) Motor cortex of the cerebrum (carnivores) and Spinal cord (herbivores)
(C) Spinal cord (both)
(D) Hippocampus (carnivores) and Purkinje cells (herbivores)

2019
87. Hyperkeratosis of the footpads and planum nasale ('hard pad disease') is a clinical and pathological manifestation of:
(A) Rabies
(B) Canine Distemper
(C) Infectious Canine Hepatitis
(D) Canine Parvovirus

2022
88. Read the following statements regarding neurotropic RNA viruses:
I. Rabies virus travels to the CNS via retrograde axoplasmic flow.
II. Canine Distemper virus causes immunosuppression by targeting lymphocytes.
III. Newcastle disease is restricted solely to the nervous system in birds.
IV. Negri bodies are composed of viral ribonucleoprotein.
V. Demyelination in distemper is partly immune-mediated.
Which of the above statements is/are incorrect?
(A) I and II
(B) III
(C) IV and V
(D) II and III

NODIA

2009
89. The inclusion bodies seen in Canine Distemper infections are uniquely:
(A) Exclusively intranuclear
(B) Exclusively intracytoplasmic
(C) Absent within host epithelial cells
(D) Intranuclear and intracytoplasmic

2006
90. Dermatophytosis (Ringworm) specifically involves fungal invasion of:
(A) Keratinized structures like hair and nails
(B) The dermis and subcutaneous adipose tissue
(C) The basal proliferating layer of the epidermis
(D) The underlying skeletal muscle

2023
91. The primary histopathological change observed in the epidermis during a dermatophyte infection is:
(A) Severe coagulative necrosis of the epidermal layer
(B) Hyperkeratosis and folliculitis with hyphae
(C) Formation of deep chronic granulomas in

2021

71. Peste des Petits Ruminants (PPR) is a paramyxovirus infection causing severe lesions primarily in which two systems?
(A) Central nervous and reproductive systems
(B) Respiratory and digestive systems
(C) Musculoskeletal and integumentary systems
(D) Cardiovascular and renal systems

2002

72. In young calves, FMD virus can cause acute mortality due to:
(A) Severe vesicular stomatitis preventing ingestion of feed
(B) Extensive acute pulmonary edema and alveolar congestion
(C) Myocardial degeneration and necrosis
(D) Spontaneous rupture of the splenic parenchyma

2017

73. The characteristic macroscopic intestinal lesion in Classical Swine Fever (Pestivirus) is:
(A) Diffuse hemorrhagic enteritis
(B) Fibrinous cast formation in the ileum
(C) Villous atrophy in the jejunum
(D) Button ulcers in the cecum and colon

2012

74. Classical Swine Fever causes generalized petechial hemorrhages primarily due to its tropism for:
(A) Endothelial cells
(B) Mucosal epithelial cells
(C) Mature hepatocytes
(D) Central neurons

2015

75. Rotaviral enteritis in neonates is characterized microscopically by:
(A) Necrosis of the intestinal crypt cells
(B) Atrophy and blunting of the apical villi
(C) Deep ulcerative lesions reaching the muscularis mucosae
(D) Granulomatous inflammation of the lamina propria

2006

76. Read the following statements regarding systemic RNA viral infections:
I. FMD virus causes vesicular lesions in pigs, cattle, and sheep.
II. PPR virus typically produces syncytia and both intranuclear and intracytoplasmic inclusions.
III. Rotavirus targets mature enterocytes at the tips of the villi.
IV. Classical Swine Fever pathologically induces severe splenomegaly without splenic infarcts.
Which of the above statements are correct?
(A) I, II and III
(B) I, III and IV
(C) II and IV
(D) I and II

2001

77. Which histological feature is highly characteristic of the lesions found in the oral cavity of animals with FMD?
(A) Spongiosis and ballooning degeneration
(B) Massive eosinophilic infiltration in the dermis
(C) Hyperkeratosis with parakeratosis
(D) Proliferation of basal cells forming a papilloma

2002

78. In PPR, typical macroscopic findings in the large intestine include:
(A) Button ulcers scattered throughout the cecum
(B) Diffuse caseous necrosis of the mucosa
(C) Zebra striping in the colon and rectum
(D) Large pedunculated polyps

2010

79. The non-suppurative encephalitis occasionally observed in Classical Swine Fever is characterized by:
(A) Massive neutrophil infiltration
(B) Formation of large necrotic abscesses
(C) Purulent exudate in the subarachnoid space
(D) Perivascular cuffing and glial nodules

2011

80. The pathognomonic histological lesion for Rabies in the central nervous system is:
(A) Cowdry Type A intranuclear inclusions in astrocytes
(B) Extensive suppurative meningoencephalitis
(C) Eosinophilic intracytoplasmic Negri bodies
(D) Spongiform vacuolation of the neuropil

2011

81. In canine distemper, the virus exhibits tropism for multiple tissues. Which of the following is NOT typically affected?
(A) Epithelial tissues
(B) Skeletal muscle
(C) Lymphoid tissues
(D) Nervous tissue

Click Here to Buy book on Flipkart

2004
122. In heavy Ascariasis in foals (Parascaris equorum), the most immediate life-threatening pathological consequence in the intestine is:
(A) Severe mucosal ulceration with extensive necrosis
(B) Mechanical impaction or acute intestinal rupture
(C) Profuse, watery to hemorrhagic diarrheal illness
(D) Chronic granulomatous enteritis with thick walls

2021
123. Which nematode is responsible for causing parasitic bronchitis characterized by the presence of adult worms in the airways of sheep and goats?
(A) Dictyocaulus filaria
(B) Haemonchus contortus
(C) Ostertagia ostertagi
(D) Toxocara canis

2017
124. The primary pathological finding in 'Moroccan leather' abomasum of cattle is due to:
(A) Diffuse mucosal hemorrhage caused by abomasal Haemonchus species
(B) Massive hepatic fibrosis resulting from migrating immature Fasciola
(C) Abomasal gland hyperplasia from emerging Ostertagia larvae
(D) Chronic granulomatous inflammation triggered by Mycobacterium species

2015
125. Consider the following statements regarding nematode pathology:
I. Haemonchus contortus causes severe blood loss.
II. Dictyocaulus viviparus causes parasitic bronchitis in cattle.
III. Oesophagostomum induces formation of nodules in the large intestine.
IV. Ascaris suum adult worms cause milk spots in the liver.
Which of the above statements is/are correct?
(A) I, II and IV
(B) I, III and IV
(C) I, II and III
(D) II, III and IV

2019
126. The eosinophilic granulomas associated with Toxocara canis larval migrans in incidental hosts (like humans) are primarily found in:
(A) The mucosal lining of the intestinal lumen
(B) Deep cutaneous and subcutaneous tissues
(C) The brain, eyes, and visceral organs
(D) Synovial membranes of major joint spaces

2001
127. Which nematode typically embeds its whip-like anterior end deep into the cecal mucosa, causing typhlitis?
(A) Toxocara canis
(B) Trichuris vulpis
(C) Ancylostoma caninum
(D) Strongyloides stercoralis

2013
128. Pathogenesis of Hookworm (Ancylostoma) infection is primarily characterized by:
(A) Biliary obstruction with secondary cholangiohepatitis
(B) Pulmonary granulomas secondary to larval migration
(C) Compressive atrophy of cerebral and cerebellar tissues
(D) Intestinal mucosal damage and acute blood loss anemia

NODIA

2022
129. The acute phase of Fasciolosis in sheep is pathologically characterized by:
(A) Traumatic hepatitis, hemorrhagic tracts, and sudden death
(B) Chronic cholangitis with extensive calcification of bile ducts
(C) Diffuse hepatic fibrosis leading to end-stage liver cirrhosis
(D) Intestinal mucosal ulceration with secondary bacterial enteritis

2017
130. Chronic Fasciola hepatica infection in cattle typically leads to:
(A) Massive hepatic necrosis with extensive parenchummorrhage
(B) Hydatid cyst formation within the left hepatic lobe parenchyma
(C) Biliary hyperplasia and periductal fibrosis ('pipe-stem' liver)
(D) Acute splenic rupture accompanied by severe hemoabdomen

2025
131. The space-occupying lesion known as a hydatid cyst is the intermediate stage of which cestode?
(A) Echinococcus granulosus
(B) Taenia solium
(C) Moniezia expansa
(D) Dipylidium caninum

tissues
(D) Diffuse melanocyte proliferation in the basal stratum

2017
92. Candidiasis, caused by Candida albicans, frequently causes opportunistic infections of the upper gastrointestinal tract in birds, leading to:
(A) Hemorrhagic enteritis
(B) Thickened, grayish-white crop pseudomembrane
(C) Deep perforating ulcers in the proventricular mucosa
(D) Caseous nodules in the liver

2015
93. Aspergillosis in avian species primarily targets the respiratory system. The typical macroscopic lesion is:
(A) Caseous nodules in the lungs and air sacs
(B) Fibrinous pleuritis without nodule formation
(C) Diffuse pulmonary hemorrhage
(D) Severe mucoid tracheitis

2002
94. Histologically, Aspergillus spp. can be identified in tissue sections by the presence of:
(A) Broad, aseptate hyphae branching at right angles in tissues
(B) Small, oval intracellular yeast cells within macrophages
(C) Narrow, septate hyphae branching at acute angles
(D) Large, thick-walled spherules containing endospores

2024
95. Match the fungal agent in List I with its characteristic pathological finding in List II.
List I: (Fungal Agent)
P. Microsporum canis
Q. Candida albicans
R. Aspergillus fumigatus
S. Trichophyton verrucosum
List II: (Pathological Finding)
1. Mycotic abortion with thickened placenta in cattle
2. Thrush (pseudomembranous stomatitis/ ingluvitis)
3. Feline ringworm
4. Bovine ringworm
(A) P-3, Q-2, R-1, S-4
(B) P-4, Q-2, R-3, S-1
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-2, S-3

2025
96. Which superficial fungal infection is characterized by round, alopecic, scaly lesions primarily affecting the head and neck region of calves?
(A) Candidiasis
(B) Cryptococcosis
(C) Ringworm
(D) Histoplasmosis

2002
97. In tissue damage caused by Candidiasis, the fungus typically presents morphologically as:
(A) Exclusively branching septate hyphae
(B) Yeast cells and pseudohyphae
(C) Exclusively budding yeast cells
(D) Arthrospores

2004
98. Brooder pneumonia in chicks is a common colloquial term for an outbreak of:
(A) Candidiasis
(B) Cryptococcosis
(C) Blastomycosis
(D) Aspergillosis

NODIA

2012
99. The inflammatory response elicited by Aspergillus hyphae invading tissues is predominantly:
(A) Granulomatous
(B) Suppurative exudate
(C) Lymphocytic response
(D) Serous inflammation

2002
100. Systemic mycotic infections typically induce which primary type of inflammatory reaction in host tissues?
(A) Purulent
(B) Serofibrinous exudation
(C) Granulomatous
(D) Catarrhal

2015
101. Histoplasmosis (caused by Histoplasma capsulatum) is pathologically characterized by the presence of the organism primarily within:
(A) Macrophages
(B) Erythrocytes
(C) Neutrophils
(D) Epithelial cells

2003
102. Read the following statements regarding systemic dimorphic fungi:
I. They exist as molds in the environment and yeasts in the host tissue.
II. Cryptococcus neoformans is characterized

by a thick polysaccharide capsule.
III. Blastomyces dermatitidis replicates by multiple budding.
Which of the above statements are correct?
(A) I and II
(B) II and III
(C) I
(D) II

2001
103. The characteristic histological appearance of Blastomyces dermatitidis in tissue sections is:
(A) Small intracellular yeasts without a capsule
(B) Large, broad-based budding yeasts
(C) Spherules containing numerous endospores
(D) Septate branching hyphae

2014
104. Cryptococcosis in cats frequently presents as gelatinous masses in the nasal cavity or central nervous system. The gelatinous appearance is due to:
(A) Extensive mucinous degeneration of host tissue
(B) Massive accumulation of eosinophils
(C) The thick polysaccharide yeast capsule
(D) Fibrinate exudation

2001
105. Due to its unique structure, the capsule of Cryptococcus neoformans in tissue sections is best visualized using which special stain?
(A) Gram stain
(B) Ziehl-Neelsen stain
(C) Hematoxylin and Eosin
(D) Mucicarmine stain

2024
106. In canine histoplasmosis, widespread involvement of the gastrointestinal tract commonly leads to:
(A) Deep, perforating hemorrhagic ulcers with transmural necrosis
(B) Thickened, corrugated mucosa with massive macrophage infiltration
(C) Diffuse mucosal villous atrophy with minimal cellular infiltration
(D) Acute intussusception accompanied by localized vascular congestion

2007
107. Cutaneous lesions in Blastomycosis are typically described as:
(A) Ulcerated, draining pyogranulomatous skin nodules
(B) Diffuse, thin crusty scales with generalized hair loss
(C) Large, non-inflammatory subcutaneous lipoma-like masses
(D) Small, painful vesicles containing serosanguinous fluid

2023
108. The 'dimorphic' nature of Blastomyces dermatitidis means that at body temperature (37°C) it grows as:
(A) A mold
(B) A hyphal mat
(C) A yeast
(D) A spherule

2023
109. Mechanical obstruction as a mechanism of parasitic tissue injury is classic for heavy infections with:
(A) Babesia bigemina within host erythrocytes
(B) Toxoplasma gondii within tissue macrophages
(C) Trichostrongylus colubriformis in the gut
(D) Ascaris suum within the small intestine

2009
110. Compressive atrophy of host tissues is the primary pathogenic mechanism for which space-occupying parasitic stage?
(A) Adult stages of Haemonchus contortus
(B) Mature Fasciola hepatica in bile ducts
(C) Echinococcus granulosus hydatid cysts
(D) Extracellular Trypanosoma evansi in blood

2003
111. Tissue migration tracts characterized by necrosis, hemorrhage, and subsequent fibrosis are hallmarks of infection by:
(A) Adult tapeworms attached to the intestinal mucosa
(B) Migrating Fasciola hepatica or Ascaris suum larvae
(C) Intracellular coccidia within intestinal epithelium
(D) Ectoparasitic mites feeding on epidermal tissues

2021
112. Nutrient competition is a significant mechanism of host injury in infections with large numbers of:
(A) Hematophagous ectoparasitic tick species
(B) Actively replicating intracellular protozoa
(C) Metrastrongyloid lungworms in host airways
(D) Adult intestinal cestodes such as Moniezia

Downloaded from www.pdf.tube

2018

113. Parasite-induced hypersensitivity reactions in the host often result in localized tissue infiltration by:
(A) Eosinophils
(B) Neutrophils
(C) Erythrocytes
(D) Fibroblasts

2003

114. The 'milk spots' observed on the liver of pigs are an example of tissue reaction to:
(A) Compressive atrophy of hepatocytes from adjacent cysts
(B) Mechanical obstruction of major intrahepatic bile ducts
(C) Focal fibrosis from migrating Ascaris suum larvae
(D) Systemic toxins released by mature adult nematodes

2018

115. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option.
Assertion (A): Heavy infections with adult Haemonchus contortus cause severe anemia and hypoproteinemia.
Reason (R): Haemonchus contortus is a voracious blood-sucking nematode residing in the abomasum.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2023

116. Which mechanism describes the tissue damage caused by strongyle larvae in the mesenteric arteries of horses?
(A) Direct nutrient competition with the host for essential dietary elements
(B) Formation of large, fluid-filled space-occupying larval cysts in organs
(C) Intracellular replication within macrophages causing acute cellular lysis
(D) Endothelial injury leading to arteritis, thrombosis, and infarction

2009

117. Anaphylactic shock can occasionally occur when which parasitic structure ruptures, releasing massive amounts of foreign antigen?
(A) Ascaris egg
(B) Hydatid cyst
(C) Coccidian oocyst
(D) Fasciola adult

2005

118. Eosinophilic granulomas in parasitic infections are formed primarily to:
(A) Digest intact, mature adult nematodes in tissues
(B) Repair mechanically damaged host mucosal epithelium
(C) Wall off and destroy parasite eggs or trapped larvae
(D) Provide a nutrient-rich microenvironment for parasites

2024

119. Haemonchosis in sheep primarily leads to which constellation of pathological findings?
(A) Severe hemorrhagic enteritis, mucosal necrosis, and perforation
(B) Granulomatous pneumonia, alveolar emphysema, and pleuritis
(C) Diffuse hepatic fibrosis, biliary hyperplasia, and ascites
(D) Severe anemia, abomasal mucosal damage, and peripheral edema

NODIA

2007

120. Dictyocaulosis (lungworm infection) in cattle primarily causes pathology in the:
(A) Bronchi and bronchioles, causing catarrhal bronchitis
(B) Alveolar parenchyma, leading to diffuse lipid pneumonia
(C) Pleural space, resulting in severe fibrinous pleuritis
(D) Tracheal epithelium, causing localized mucosal necrosis

2018

121. The macroscopic presence of small, white, raised nodules in the intestinal wall of sheep is commonly referred to as 'pimply gut' and is caused by:
(A) The abomasal nematode Haemonchus contortus
(B) The nodular worm Oesophagostomum columbianum
(C) The cecal whipworm nematode Trichuris ovis
(D) The trematode liver fluke Fasciola hepatica

basal cell layer
(D) Neoplastic transformation and proliferation of dermal melanocytes

2023
61. Infectious Laryngotracheitis (ILT) in poultry is caused by a Herpesvirus. The pathognomonic lesion observed in the trachea is:
(A) Hemorrhagic tracheitis with intranuclear inclusions
(B) Catarrhal tracheitis with intracytoplasmic inclusions
(C) Granulomatous tracheitis with multi-nucleated giant cells
(D) Fibrous stricture with marked narrowing of tracheal lumen

2025
62. Marek's Disease, a herpesviral infection in chickens, is pathologically characterized by:
(A) Neoplastic transformation of B-lymphocytes within the bursa of Fabricius
(B) Acute necrotizing inflammation of the mucosal intestinal epithelium
(C) Neoplastic T-lymphocyte infiltration in nerves and organs
(D) Chronic suppurative inflammatory reactions within the joint spaces

2004
63. Adenoviral infections in dogs, specifically Infectious Canine Hepatitis (CAV-1), predominantly cause lesions in the:
(A) Heart and skeletal muscle
(B) Respiratory epithelium and lungs
(C) Intestinal mucosa
(D) Liver and vascular endothelium

2022
64. Parvovirus primarily targets rapidly dividing cells. In young puppies, this results in:
(A) Suppurative meningoencephalitis of the cerebral cortex
(B) Diffuse granulomatous pneumonia of the pulmonary lobes
(C) Necrotizing enteritis and myocarditis
(D) Acute necrotizing renal tubular necrosis

2008
65. Read the given assertion and reason, and select the correct option.
Assertion (A): Parvoviral enteritis results in severe villous atrophy and crypt necrosis.
Reason (R): Parvoviruses require cells in the S-phase of the cell cycle for replication, and intestinal crypt epithelium is highly mitotically active.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2023
66. The macroscopic appearance of 'classic' fowl pox involves:
(A) Hemorrhagic ulcers in the proventriculus
(B) Swollen, hemorrhagic bursa of Fabricius
(C) Diffuse enlargement of the sciatic nerve
(D) Wart-like, scabby skin lesions

2025
67. The intranuclear inclusion bodies characteristic of Infectious Canine Hepatitis are known as:
(A) Cowdry Type A
(B) Negri bodies
(C) Bolinger bodies
(D) Guarnieri bodies

2012
68. In Marek's Disease, classical macroscopic examination often reveals:
(A) Extensive caseous nodules within the pulmonary parenchyma
(B) Severe acute hemorrhagic enteritis of the small intestine
(C) Thickening and loss of striation in peripheral nerves
(D) Diffuse fibrinous pericarditis and associated perihepatitis

2016
69. Canine parvovirus enters the host primarily via the oronasal route and first replicates in which tissue?
(A) Oropharyngeal lymphoid tissue
(B) Cardiac myofibers and myocardium
(C) Replicating intestinal crypt cells
(D) Hematopoietic cells in bone marrow

2014
70. Foot-and-Mouth Disease (FMD) is pathologically defined by the presence of:
(A) Chronic granulomas in the lungs and liver
(B) Hemorrhagic enteritis and colitis
(C) Demyelinating encephalitis
(D) Vesicles on oral mucosa and feet

Click Here to Buy book on Amazon

2006
132. Cysticercus bovis, the intermediate stage of Taenia saginata, produces lesions commonly found in the:
(A) Hepatic and pulmonary parenchyma of the liver and lungs
(B) Central nervous system tissues including brain and spinal cord
(C) Mucosal and submucosal layers of the intestinal tract
(D) Striated muscles including the heart, masseter, and tongue

2025
133. Neurocysticercosis in humans and pigs is caused by the larval stage of:
(A) Taenia saginata
(B) Taenia solium
(C) Echinococcus multilocularis
(D) Fasciola hepatica

2004
134. Which mechanism leads to the extensive biliary hyperplasia observed in chronic fasciolosis?
(A) Compression of the bile ducts by adjacent granulomatous reactions
(B) Direct intracellular replication of flukes within biliary epithelium
(C) Mechanical irritation by flukes and toxic excretion products
(D) Systemic immune-complex deposition within the major duct walls

2019
135. Match the parasite in List I with its corresponding characteristic lesion in List II.
List I: (Parasite)
P. Fasciola hepatica
Q. Echinococcus granulosus
R. Taenia solium (larvae)
S. Moniezia expansa
List II: (Characteristic Lesion)
1. Hydatid cyst in liver/lungs
2. Measly pork (Cysticercosis)
3. Pipe-stem liver
4. Minimal pathology, possible nutrient competition in sheep gut
(A) P-3, Q-1, R-2, S-4
(B) P-1, Q-3, R-2, S-4
(C) P-3, Q-2, R-1, S-4
(D) P-2, Q-1, R-4, S-3

2019
136. The fluid inside a fertile hydatid cyst contains which infective structures?
(A) Protoscoleces
(B) Metacercariae
(C) Miracidia
(D) Coracidia

2011
137. Which of the following best describes the 'measles' lesion seen in Cysticercosis?
(A) A large, unilocular fluid-filled cyst with a thick germinal layer
(B) A small, oval vesicle containing a single invaginated scolex
(C) A solid, caseous granuloma
(D) A diffuse network of fibrous tissue

2021
138. The primary pathological finding in heavy paramphistomosis in ruminants is caused by:
(A) Adult flukes residing in the rumen causing severe papillar necrosis
(B) Duodenal migration of immature flukes causing hemorrhagic enteritis
(C) Adult flukes localized within the bile ducts causing severe cholangitis
(D) Immature larval flukes aberrantly encysting in the cerebral cortex

2003
139. Intravascular hemolysis, hemoglobinuria, and severe anemia are hallmark pathological features of infection by:
(A) Babesia bovis
(B) Theileria parva
(C) Trypanosoma evansi
(D) Toxoplasma gondii

NODIA

2010
140. Which of the following lesions is commonly observed in the kidneys during acute bovine babesiosis?
(A) Severe interstitial nephritis with focal granuloma formation
(B) Massive renal cortical necrosis with extensive hemorrhages
(C) Dark red to black kidneys from hemoglobin cast formation
(D) Suppurative pyelonephritis with ascending bacterial infection

2024
141. The pathogenesis of 'cerebral babesiosis' caused by Babesia bovis is primarily due to:
(A) Direct destruction of neurons by replicating protozoa
(B) Extensive hemorrhage from ruptured major cerebral arteries
(C) Acute suppurative meningoencephalitis with

microabscesses
(D) Aggregation of infected erythrocytes in brain capillaries

2023
142. Theileriosis (East Coast Fever) caused by Theileria parva primarily targets which host cells before entering erythrocytes?
(A) Hepatocytes
(B) Endothelial cells
(C) Lymphocytes
(D) Neutrophils

2006
143. The characteristic microscopic structures seen in the cytoplasm of lymphocytes infected with Theileria are called:
(A) Neuronal Negri inclusion bodies
(B) Koch's blue bodies (schizonts)
(C) Erythrocytic Howell-Jolly bodies
(D) Oxidized erythrocyte Heinz bodies

2011
144. Consider the following statements regarding hemoprotozoan pathology:
I. Babesia causes massive intravascular hemolysis.
II. Theileria annulata causes severe lymphoproliferative disease followed by lymphoid depletion.
III. Trypanosomiasis consistently causes acute intravascular hemolysis.
IV. Icterus is a common clinical sign in acute babesiosis.
V. Babesia bigemina is transmitted by ixodid ticks.
Which of the above statements is/are INCORRECT?
(A) I and II
(B) II and IV
(C) III
(D) I and V

2015
145. Trypanosomiasis (e.g., Surra) pathogenesis is heavily influenced by the parasite's ability to:
(A) Undergo antigenic variation of its surface glycoproteins
(B) Replicate rapidly within host erythrocytes causing cellular lysis
(C) Form large, space-occupying parasitic cysts in the liver parenchyma
(D) Produce potent systemic endotoxins causing severe neurological damage

2001
146. In acute cases of Babesiosis, the spleen typically appears:
(A) Severely contracted, pale, and chronically fibrotic
(B) Completely normal in size with prominent lymphoid follicles
(C) Markedly enlarged, dark, and soft (splenomegaly)
(D) Diffusely riddled with multiple caseous abscesses

2016
147. Anemia in Trypanosomiasis is primarily considered to be:
(A) Extravascular hemolytic and dyshemopoietic
(B) Intravascular hemolytic
(C) Hemorrhagic due to massive blood loss
(D) Iron deficiency due to parasite consumption

2024
148. The profound generalized lymphadenopathy seen in the early stages of East Coast Fever (Theileriosis) is due to:
(A) Infiltration of neutrophils in response to bacterial co-infection
(B) Caseous necrosis of the medullary cords
(C) Fibrous tissue replacement of the cortex
(D) Massive proliferation of parasitized lymphocytes

2007
149. Coccidiosis in poultry, caused by Eimeria tenella, specifically localizes in the:
(A) Crop
(B) Ceca
(C) Duodenum
(D) Proventriculus

2019
150. The primary mechanism of epithelial necrosis and hemorrhage in intestinal coccidiosis is:
(A) Release of toxic bacterial endotoxins by secondary invaders
(B) Compressive atrophy of cells from large subepithelial cysts
(C) Rupture of enterocytes during release of merozoites
(D) Autoimmune-mediated destruction of host intestinal villi

2003
151. Toxoplasmosis in pregnant ewes classically results in abortion. The characteristic macroscopic lesion on the placenta is:
(A) Leathery thickening of intercotyledonary

Downloaded from www.pdf.tube

areas
(B) Diffuse hemorrhagic placentitis with fetal edema
(C) Purulent exudate covering the chorionic membrane
(D) Focal necrosis and calcification of cotyledons

2025
152. Histopathologically, Neosporosis in aborted bovine fetuses is most consistently characterized by:
(A) Non-suppurative encephalomyelitis with focal necrosis
(B) Diffuse pulmonary edema and extensive alveolar congestion
(C) Severe suppurative enteritis with mucosal ulceration
(D) Fibrinous epicarditis and mild localized myocarditis

2005
153. Which of the following enteric protozoa resides primarily attached to the brush border of enterocytes, causing malabsorptive diarrhea without deep invasion?
(A) Eimeria bovis
(B) Cryptosporidium parvum
(C) Toxoplasma gondii
(D) Sarcocystis cruzi

2013
154. Sarcocystis spp. form highly organized cysts primarily within which host tissue?
(A) Mucosal epithelium of the intestinal tract
(B) Striated skeletal and cardiac musculature
(C) Parenchyma of the central nervous system
(D) Deep parenchyma of the host liver tissue

2015
155. Consider the following statements regarding tissue protozoa:
I. Toxoplasma gondii definitive hosts are felines.
II. Neospora caninum is a major cause of abortion in cattle.
III. Eimeria stiedae causes hepatic coccidiosis in rabbits.
IV. Sarcocystis cysts in meat are typically microscopic or macroscopic, depending on the species.
V. Cryptosporidium invades the deep lamina propria.
Which of the above statements is/are correct?
(A) I, II, III and IV
(B) I, II, IV and V
(C) I, III, IV and V
(D) II, III, IV and V

2018
156. Hepatic coccidiosis in rabbits is characterized by:
(A) Severe hepatic lipidosis with extensive vacuolar change
(B) Massive centrilobular coagulative necrosis of hepatocytes
(C) Enlarged bile ducts with papillary hyperplasia and oocysts
(D) Disseminated granulomas throughout the hepatic parenchyma

2007
157. The tissue cysts (bradyzoites) of Toxoplasma gondii are most commonly found in:
(A) Circulating erythrocytes and leukocytes
(B) Mucosal lining of the intestinal lumen
(C) Epidermal layer of skin and hair follicles
(D) Central nervous system and skeletal muscle

NODIA

2007
158. In neonatal calves, Cryptosporidiosis primarily leads to which intestinal pathological change?
(A) Deep necrotic ulcers extending to the serosa
(B) Massive hemorrhage into the lumen
(C) Mild to moderate villous atrophy and fusion
(D) Granulomatous inflammation of the crypts

2024
159. Contagious Bovine Pleuropneumonia (CBPP) is caused by Mycoplasma mycoides subsp. mycoides. The pathognomonic gross lesion is:
(A) Bilateral purulent bronchopneumonia with abscess formation
(B) Fibrinous bronchopneumonia with marbling and pleuritis
(C) Diffuse interstitial pneumonia with alveolar septal damage
(D) Granulomatous pneumonia with central caseous necrosis

2002
160. The characteristic 'marbling' of the lungs in CBPP is primarily due to:
(A) Alternate areas of pale and dark caseous necrosis in parenchyma
(B) Extensive deposition of melanin pigment in the alveolar spaces
(C) Parasitic migration tracts crisscrossing the major pulmonary lobes
(D) Interlobular septal edema and variable lobular consolidation

(C) Morula
(D) Bradyzoite

2011

180. The metabolically active, intracellular replicative form of Chlamydia is the:
(A) Elementary body
(B) Tachyzoite
(C) Spore
(D) Reticulate body

2004

181. Enzootic abortion of ewes (EAE), caused by Chlamydia abortus, is pathologically characterized by:
(A) Minimal placental changes accompanied by severe fetal malformations
(B) Necrotizing placentitis of cotyledons and intercotyledonary areas
(C) Acute hemorrhagic metritis accompanied by massive maternal blood loss
(D) Chronic granulomatous lesions restricted strictly to maternal ovaries

2001

182. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option.
Assertion (A): Avian chlamydiosis (Psittacosis) often presents with severe fibrinous polyserositis.
Reason (R): Chlamydia psittaci causes systemic vascular damage, leading to the exudation of fibrin-rich fluid into body cavities.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2025

183. Macroscopic examination of a bird with severe Psittacosis typically reveals:
(A) Severe hemorrhagic tracheitis with caseous bronchial plugs
(B) Diffuse caseous nodules throughout the host intestinal wall
(C) Extensive neoplastic lymphoid tumors distributed in the liver
(D) Marked splenomegaly, hepatomegaly, and fibrinous pericarditis

2018

184. In tissue sections, Chlamydial microcolonies can be identified within the cytoplasm of host cells. These are visible using which stain?
(A) Modified Ziehl-Neelsen or Gimenez stain method
(B) Grocott's methenamine silver histochemical stain
(C) Congo Red amyloid identification stain
(D) Fontana-Masson silver-based melanin stain

2016

185. Select the correct sequence of the Chlamydial developmental cycle:
(A) Attachment of EB → Internalization → Conversion to RB → Multiplication of RB → Conversion to EB → Cell lysis and release
(B) Attachment of RB → Internalization → Conversion to EB → Multiplication of EB → Conversion to RB → Cell lysis and release
(C) Internalization of EB → Multiplication of EB → Conversion to RB → Cell lysis and release
(D) Attachment of RB → Multiplication of RB → Conversion to EB → Cell lysis and release

2001

186. Apart from abortion, Chlamydial infections in ruminants can also manifest pathologically as:
(A) Severe vesicular oral stomatitis
(B) Polyarthritis and conjunctivitis
(C) Chronic spongiform encephalopathy
(D) Demyelinating peripheral neuropathy

2010

187. The placental lesions in EAE (Chlamydia abortus) most closely resemble those caused by:
(A) Brucella abortus
(B) Campylobacter fetus
(C) Neospora caninum
(D) Listeria monocytogenes

2003

188. In feline hosts, Chlamydia felis is best known for causing which primary pathological condition?
(A) Chronic progressive interstitial renal failure
(B) Acute necrotizing lymphocytic enteritis
(C) Severe bilateral conjunctivitis and rhinitis
(D) Diffuse interstitial eosinophilic pneumonia

2017

189. The pathogenesis of prion diseases fundamentally involves:
(A) Transcription of viral RNA within host brain neurons
(B) Direct bacterial production of systemic neurotoxins
(C) Chronic immune-complex deposition in the choroid plexus
(D) Conformational change of a normal cellular

Click Here to Buy book on Flipkart

(D) Acute dilation and hypertrophy of the right ventricle

2024

171. Anaplasma marginale causes severe disease in cattle primarily through:
(A) Replication within red blood cells causing extravascular hemolysis
(B) Extensive damage to vascular endothelium resulting in hemorrhage
(C) Secretion of potent circulating neurotoxins causing paralysis
(D) Disseminated granulomatous inflammatory lesions in the lung tissue

2018

172. The hallmark lesion of canine ehrlichiosis (Ehrlichia canis) in the acute phase is:
(A) Severe suppurative arthritis of major articular joints
(B) Caseous lymphadenitis of superficial lymph node chains
(C) Widespread vasculitis, hemorrhage, and pancytopenia
(D) Acute necrotizing pancreatitis with parenchymal fat necrosis

2023

173. Microscopic examination of brain smears (e.g., from the hippocampus) in cases of Heartwater reveals:
(A) Eosinophilic Negri inclusion bodies within cortical neurons
(B) Extensive spongiform vacuolation within the brain neuropil
(C) Large colonies of extracellular bacteria in cerebral vessels
(D) Morulae clusters in the cytoplasm of endothelial cells

2022

174. Which of the following is a classic clinical and pathological feature of Anaplasmosis, distinguishing it from Babesiosis?
(A) Presence of severe, acute-onset hemoglobinuria
(B) Profound central nervous system demyelinating lesions
(C) Absence of hemoglobinuria despite severe anemia
(D) Severe hemorrhagic enteritis of the small intestine

2002

175. Match the Rickettsial agent in List I with its primary target cell in List II.

List I: (Rickettsial Agent)
P. Anaplasma marginale
Q. Ehrlichia canis
R. Ehrlichia ruminantium
S. Anaplasma platys

List II: (Target Cell)
1. Erythrocytes
2. Monocytes/Macrophages
3. Endothelial cells
4. Platelets

(A) P-1, Q-2, R-3, S-4
(B) P-2, Q-1, R-4, S-3
(C) P-1, Q-3, R-2, S-4
(D) P-3, Q-2, R-1, S-4

2023

176. The pathogenesis of tick-borne fever caused by Anaplasma phagocytophilum involves infection of:
(A) Mature host erythrocytes
(B) Vascular endothelial cells
(C) Replicating host lymphocytes
(D) Neutrophils and eosinophils

2025

177. The generalized edema (including pulmonary edema and hydrothorax) seen in Heartwater is a direct consequence of:
(A) Endothelial damage causing increased vascular permeability
(B) Left-sided congestive heart failure with alveolar congestion
(C) Severe systemic hypoproteinemia resulting from liver failure
(D) Chronic renal glomerular amyloidosis with protein-losing nephropathy

2005

178. Erythrocyte destruction in Anaplasmosis is primarily mediated by:
(A) Direct cell lysis during the active release of new parasites
(B) Secretion of potent bacterial hemolysins into systemic plasma
(C) Phagocytosis of infected, opsonized red cells by macrophages
(D) Mechanical shear stress experienced in narrow host capillaries

2002

179. Chlamydial organisms exhibit a unique biphasic developmental cycle. The infectious, extracellular form is known as the:
(A) Elementary body
(B) Reticulate body

Downloaded from www.pdf.tube

Leukocidins destroy white blood cells, reducing host defense. Collagenase degrades structural proteins in connective tissues, facilitating tissue invasion. Together, these virulence factors allow pathogens to overcome host physical barriers and immune responses, promoting colonization and dissemination.

19. (A) IL-1 and TNF-alpha
Tissue injury and immune activation by bacterial endotoxins lead to the massive release of primary pro-inflammatory cytokines, specifically tumor necrosis factor-alpha and interleukin-1. Produced largely by activated macrophages, these signaling molecules drive systemic responses including fever, vascular endothelial activation, increased capillary permeability, and leukocytosis, which can progress to lethal endotoxic shock.

20. (C) Disruption of cyclic nucleotide pathways causing fluid secretion
Enterotoxigenic pathogens, such as enterotoxigenic Escherichia coli, cause tissue injury and watery diarrhea by producing enterotoxins that bind to mucosal receptors. This binding permanently activates adenylate or guanylate cyclase, elevating intracellular cyclic AMP or GMP levels. High cyclic nucleotide levels inhibit sodium absorption and stimulate active chloride secretion, driving massive osmotic fluid loss without causing structural cell necrosis.

21. (A) Presence of specific host cell surface receptors
Cellular tropism refers to the specificity of a virus for infecting particular cell types within a host. This preference is primarily dictated by the interaction between viral attachment proteins and specific surface receptors on the host cell. Without these matching receptors, the virus cannot attach, enter, or replicate within the cell, restricting its infectivity.

22. (D) Degeneration, swelling, and lysis
Direct viral cytopathic effects are morphological alterations in host cells caused directly by viral replication. Microscopically, these changes typically present as cell degeneration, cellular swelling from altered membrane permeability, and eventual lysis as mature virions are released. These events disrupt cellular homeostasis and lead to cell death, contributing directly to tissue damage and clinical pathology.

23. (B) Fusion of adjacent cell membranes into multinucleated giant cells
Syncytia formation is a distinctive cytopathic effect where infected host cells fuse with neighboring uninfected cells, creating large, multinucleated giant cells. This fusion is mediated by viral envelope glycoproteins expressed on the surface of the infected cell. This process allows the virus to spread directly from cell to cell, effectively evading circulating neutralizing antibodies.

24. (A) I, II and IV
Viral inclusion bodies are intracellular structures that represent active sites of viral replication, transcription, or virion assembly. They can be located in either the cytoplasm or the nucleus and are easily identified using routine light microscopy. Since both DNA and RNA viruses can produce these structures, they serve as crucial diagnostic hallmarks in histopathology.

NODIA

25. (D) Rabies virus
Rabies virus infection is characterized by the presence of Negri bodies, which are highly specific, eosinophilic, intracytoplasmic inclusion bodies found in infected neurons. These structures represent sites of active viral transcription and assembly. They are most commonly observed in the Purkinje cells of the cerebellum in herbivores and the pyramidal cells of the hippocampus in carnivores.

26. (A) Direct lysis of target lymphoid cells
Many viruses induce severe immunosuppression by directly infecting and lysing target lymphoid cells, such as lymphocytes or macrophages. This direct cytolytic destruction depletes key immune cell populations in organs like the thymus, lymph nodes, and spleen. The loss of these essential immune cells severely compromises host defense, leaving the animal highly vulnerable to secondary opportunistic infections.

27. (B) Herpesviridae
Herpesvirus infections are characterized by the formation of distinct intranuclear inclusion bodies, historically called Cowdry type A, which often exhibit an owl's eye appearance. These large, eosinophilic structures are surrounded by a clear halo within the nucleus. They represent sites of viral capsid assembly and are major diagnostic hallmarks of herpesviral pathology in tissue sections.

protein

2016

190. Consider the following statements regarding the pathogenic prion protein ($\mathrm{Pr}\,P^{\{Sc\}}$):

I. It is rich in alpha-helical structures compared to the normal protein.
II. It is highly resistant to protease degradation.
III. It accumulates and forms amyloid plaques in the brain.

Which of the above statements is/are correct?

(A) I and II
(B) II and III
(C) I
(D) II

2009

191. Normal cellular prion protein ($\mathrm{Pr}\,P^{c}$) is predominantly composed of which secondary structure?

(A) Beta-pleated sheets
(B) Alpha-helices
(C) Random coils
(D) Disulfide bridges

2007

192. Transmissible Spongiform Encephalopathies (TSEs) are unique infectious diseases because the etiologic agent:

(A) Contains single-stranded DNA
(B) Contains double-stranded RNA
(C) Replicates exclusively in macrophages
(D) Lacks any nucleic acid genome

2005

193. The primary mode of transmission for Bovine Spongiform Encephalopathy (BSE) was determined to be:

(A) Ingestion of contaminated meat-and-bone meal
(B) Aerosol transmission from infected herd mates
(C) Vertical transmission from dams to offspring
(D) Vector-borne mechanical transmission by ticks

2013

194. Which normal physiological process is bypassed by the pathogenic prion protein, leading to its accumulation in lysosomes and subsequent cellular damage?

(A) Anaerobic glycolysis pathway
(B) Mitochondrial oxidative phosphorylation
(C) Proteosomal and lysosomal degradation
(D) Free radical induced lipid peroxidation

2022

195. Arrange the following events in the correct sequence of the pathogenesis of orally acquired TSEs:

I. Retrograde spread along autonomic nerves to the CNS
II. Accumulation in gut-associated lymphoid tissue (Peyer's patches)
III. Ingestion of the pathogenic prion
IV. Neuronal vacuolation and spongiform change in the brain

Which of the following represents the correct sequence?

(A) III, II, I, IV
(B) III, I, II, IV
(C) II, III, I, IV
(D) I, II, III, IV

2005

196. The transformation of $\mathrm{Pr}\,P^{c}$ to $\mathrm{Pr}\,P^{sc}$ is believed to occur:

(A) Inside the cell nucleus during transcription
(B) Within the inner mitochondrial membranes
(C) Exclusively within the extracellular matrix
(D) At the cell membrane or endocytic vesicles

2025

197. The hallmark histological lesion in the brain of animals with Transmissible Spongiform Encephalopathies is:

(A) Extensive infiltration of perivascular lymphocytes in cortex
(B) Neuronal vacuolation and neuropil spongiform change
(C) Suppurative meningoencephalitis with multiple microabscesses
(D) Diffuse demyelination of focal cerebral white matter tracts

2018

198. Match the TSE disease in List I with its primary host in List II.

List I: (TSE Disease)

P. Scrapie
Q. BSE
R. Chronic Wasting Disease (CWD)
S. Transmissible Mink Encephalopathy

List II: (Primary Host)

1. Cattle
2. Sheep and Goats
3. Deer and Elk
4. Mink

(A) P-2, Q-1, R-3, S-4
(B) P-1, Q-2, R-3, S-4
(C) P-2, Q-3, R-1, S-4

Click Here to Buy book on Amazon

NODIA

2021
161. Contagious Caprine Pleuropneumonia (CCPP) differs pathologically from CBPP in that CCPP typically:
(A) Lacks the pronounced interlobular septal marbling
(B) Frequently causes massive sequestra formation in lungs
(C) Confines pathology strictly to upper respiratory tracts
(D) Results primarily in severe suppurative bronchopneumonia

2004
162. Chronic Avian Respiratory Disease (CRD) is primarily caused by:
(A) Mycoplasma synoviae
(B) Pasteurella multocida
(C) Mycoplasma gallisepticum
(D) Avian Influenza Virus

2014
163. The typical pathological findings in poultry complicated CRD include:
(A) Thickened air sacs with caseous exudate
(B) Severe hemorrhagic tracheitis and dyspnea
(C) Multiple granulomatous nodules in the liver
(D) Diffuse demyelination of peripheral nerves

2024
164. Polyserositis caused by Mycoplasma species typically involves fibrinous inflammation of the:
(A) Cerebral meninges and ventricular ependyma
(B) Articular and sheath synovial membranes only
(C) Valvular and mural endocardium of the heart
(D) Pleura, pericardium, and peritoneal cavity

2010
165. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option.
Assertion (A): Mycoplasma organisms are completely resistant to beta-lactam antibiotics like penicillin.
Reason (R): Mycoplasma organisms lack a rigid peptidoglycan cell wall.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2007
166. A common sequela in the chronic stage of CBPP is the formation of a 'sequestrum'. What does this represent?
(A) Accumulation of pure purulent exudate in the pleural cavity
(B) Calcified parasitic cyst embedded in deep lung parenchyma
(C) Necrotic lung tissue walled off by a fibrous capsule
(D) Benign neoplastic nodule derived from type II alveolar cells

2006
167. Mycoplasma synoviae infection in birds primarily manifests pathologically as:
(A) Focal encephalomalacia within the cerebral cortex
(B) Acute hemorrhagic enteritis of the small intestine
(C) Progressive muscular dystrophy of skeletal muscles
(D) Synovitis with purulent joint and sheath exudate

2012
168. Which feature makes Mycoplasmas uniquely difficult to identify in routine histopathological sections using standard H&E staining?
(A) Their small size and lack of cell wall make them invisible
(B) They possess a thick, waxy, hydrophobic lipid capsule
(C) They replicate exclusively inside the nuclei of host cells
(D) They rapidly undergo complete autolysis during fixation steps

2023
169. Rickettsial organisms like Ehrlichia ruminantium (agent of Heartwater) exhibit a strong tropism for which host cell type?
(A) Hepatic parenchymal cells
(B) Vascular endothelial cells
(C) Mature host erythrocytes
(D) Mucosal gut enterocytes

2013
170. The primary gross pathological finding that gives 'Heartwater' its name is:
(A) Severe hemorrhagic necrosis of the myocardial tissue
(B) Pronounced accumulation of fluid in the pericardium
(C) Chronic fibrinous pericarditis with extensive adhesions

2. Fungal elements
3. Mycobacterium species
4. Spirochetes (e.g., Leptospira)

(A) P-3, Q-2, R-1, S-4
(B) P-2, Q-3, R-1, S-4
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-2, R-3, S-4

2001

209. Select the correct sequential order of steps in a standard indirect immunohistochemistry protocol on formalin-fixed tissue:

(A) Primary antibody incubation → Antigen retrieval → Secondary antibody incubation → Chromogen application
(B) Antigen retrieval → Primary antibody incubation → Secondary antibody incubation → Chromogen application
(C) Chromogen application → Antigen retrieval → Primary antibody incubation → Secondary antibody incubation
(D) Secondary antibody incubation → Antigen retrieval → Primary antibody incubation → Chromogen application

2002

210. The fundamental property that allows Mycobacteria to retain the primary stain (carbol fuchsin) during the Ziehl-Neelsen procedure despite acid-alcohol washing is the presence of:

(A) A thick outer peptidoglycan layer
(B) A dense carbohydrate capsule layer
(C) Mycolic acid in their cell wall
(D) Intracellular polyphosphate granules

****

Downloaded from www.pdf.tube

(D) P-3, Q-2, R-1, S-4

2022

199. Unlike many viral encephalitides, the pathological findings in TSEs conspicuously lack:
(A) Marked astrocyte hypertrophy and hyperplasia
(B) Progressive neuronal loss and degeneration
(C) Widespread neuropil spongiform vacuolation
(D) An inflammatory immune cell response

2008

200. In Scrapie, which specific anatomical region of the brain is most consistently examined histologically for diagnostic vacuolation?
(A) Medulla oblongata at the level of the obex
(B) Gray matter layers of the frontal cerebral cortex
(C) Purkinje cell layers of the cerebellar cortex
(D) Sensation pathways of the anterior olfactory bulb

2022

201. Consider the following statements regarding TSE neuropathology:
I. Scrapie causes intense pruritus and wool loss.
II. BSE lesions are predominantly inflammatory.
III. Astrogliosis (proliferation of astrocytes) is a common feature.
IV. The disease is invariably fatal.
Which of the above statements is/are INCORRECT?
(A) I and III
(B) II
(C) III and IV
(D) I and IV

2016

202. The vacuolation seen in TSEs primarily affects the:
(A) Internal nuclear membrane of the host neuron
(B) Neuronal cytoplasm (perikaryon) and neuropil
(C) Outer myelin sheath layer of the long axons
(D) Endothelial cells lining cerebral capillaries

2021

203. Immunohistochemistry (IHC) for the definitive diagnosis of TSEs relies on identifying the accumulation of:
(A) Pathogenic prion protein ($Pr P^{sc}$)
(B) Glial fibrillary acidic protein (GFAP)
(C) Negri bodies
(D) Viral capsid antigens

NODIA

2002

204. In diagnostic pathology, the Ziehl-Neelsen stain is classically used to demonstrate:
(A) Pathogenic fungal hyphae and spores
(B) Intranuclear viral inclusion bodies
(C) Acid-fast Mycobacterium bacilli
(D) Degenerative prion amyloid plaques

2015

205. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option.
Assertion (A): The Periodic Acid-Schiff (PAS) stain is highly effective for identifying fungal elements in tissue sections.
Reason (R): The PAS stain reacts with the abundant polysaccharides present in fungal cell walls, turning them a bright magenta color.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2015

206. Grocott's methenamine silver (GMS) stain highlights fungal walls by rendering them:
(A) Bright magenta red
(B) Fluorescent green
(C) Dark brown to black
(D) Deep Prussian blue

2009

207. Immunohistochemical (IHC) diagnostic techniques rely primarily on the specific binding between:
(A) Basic cationic dyes and acidic cellular components
(B) An antibody and its specific host tissue antigen
(C) Heavy silver metals and fungal wall polysaccharides
(D) Acid-fast carbol fuchsin stains and mycolic acids

2005

208. Match the special stain in List I with its target pathogen/structure in List II.
List I: (Special Stain)
P. Ziehl-Neelsen
Q. Grocott's Methenamine Silver
R. Mucicarmine
S. Warthin-Starry (silver stain)
List II: (Target Pathogen or Structure)
1. Cryptococcus capsule

9. (D) Low pH of gastric secretions
The highly acidic environment of the stomach, characterized by a low pH, is a primary physiological barrier against ingested pathogens. Most bacteria and viruses entering the gastrointestinal tract are denatured or killed by this extreme acidity. This protective mechanism limits pathogen survival and colonization, protecting downstream intestinal segments from infection by acid-sensitive microorganisms.

10. (A) Virulence
Virulence is the quantitative measure of a pathogen's capability to cause disease in a susceptible host. While pathogenicity represents the qualitative ability of an infectious agent to induce disease, virulence describes the degree or severity of that disease. It is often measured by parameters such as the infectious dose or the lethal dose in animal models.

11. (B) Lipopolysaccharide
Lipopolysaccharide, located within the outer membrane of Gram-negative bacteria, is the primary endotoxin responsible for triggering septic and endotoxic shock. Upon bacterial lysis, lipopolysaccharide is released into circulation, where it binds to immune cell receptors, inducing a massive release of pro-inflammatory cytokines. This systemic inflammatory cascade leads to vasodilation, endothelial damage, and hypotension.

12. (A) I and II
Bacterial exotoxins are highly toxic proteins synthesized and actively secreted by living, metabolically active bacteria. Because of their proteinaceous nature, exotoxins are highly immunogenic and can be treated chemically, such as with formaldehyde, to form non-toxic but immunogenic toxoids for vaccination. In contrast, endotoxins are structural lipopolysaccharide components of the outer membrane of Gram-negative bacteria.

13. (A) Hyaluronidase
Hyaluronidase is an extracellular enzyme secreted by pathogenic bacteria that degrades hyaluronic acid, a major glycosaminoglycan in the extracellular matrix of connective tissues. By cleaving this structural component, hyaluronidase decreases tissue viscosity, enabling the pathogen to spread easily through tissue planes. This enzyme is therefore commonly referred to as a spreading factor.

14. (C) Preventing phagocytosis by host immune cells
The bacterial capsule is a specialized outer layer, typically composed of polysaccharides, that serves as a vital virulence factor by inhibiting phagocytosis. It masks bacterial surface antigens, prevents the deposition of complement proteins like C3b, and reduces physical attachment by neutrophils and macrophages. This allows encapsulated pathogens to evade early host clearance and establish systemic infections.

15. (D) Production of massive amounts of endotoxin
Mycobacterium species are intracellular pathogens that survive within host macrophages by resisting lysosomal enzymes, inhibiting phagosome-lysosome fusion, or escaping into the cytoplasm. However, they do not produce endotoxins. Endotoxins are structural lipopolysaccharide components found exclusively in the outer membranes of Gram-negative bacteria, whereas Mycobacterium species are Gram-positive, acid-fast bacilli with lipid-rich walls.

16. (C) Secretion of hemolysins
Hemolysins are bacterial exotoxins that directly target and lyse host red blood cells by disrupting their cytoplasmic membranes. This lysis releases hemoglobin and intracellular iron, which the bacteria can utilize for their metabolic processes and growth. This cytolytic activity is a key virulence mechanism in many pathogenic bacteria, including Streptococcus and Staphylococcus species.

17. (B) Enterotoxins
Enterotoxins are a specific class of bacterial exotoxins that target and alter the function of mucosal epithelial cells in the intestines. These toxins disrupt cellular signaling, often leading to massive fluid and electrolyte secretion into the gut lumen, which clinically manifests as diarrhea. Unlike general cytotoxins, enterotoxins specifically compromise gastrointestinal secretory and absorptive physiology.

18. (A) P-3, Q-4, R-1, S-2
Coagulase initiates fibrin clot formation, protecting bacteria from phagocytes. Kinases dissolve these clots to let bacteria spread.

Click Here to Buy book on Flipkart

ANSWERS

1. (B) The mere presence of bacteria in the bloodstream without active multiplication
Bacteremia refers specifically to the transient or passive presence of viable bacteria circulating within the bloodstream without active replication. This condition is distinct from septicemia, where bacteria actively multiply in circulation and release toxins, initiating systemic inflammatory responses and clinical signs. Bacteremia can occur after minor trauma, dental procedures, or localized infections.

2. (D) Gastrointestinal tract
Enteritis involves inflammation of the small intestine, making the gastrointestinal tract the primary route of entry for pathogens causing this condition. Infectious agents, including various bacteria, viruses, and parasites, are typically ingested via contaminated food or water. These pathogens survive the gastric acid barrier to colonize and damage the intestinal mucosal lining, leading to clinical disease.

3. (A) The pathogen actively multiplies within the circulatory system causing systemic illness
Septicemia represents a severe systemic disease characterized by the active multiplication of pathogenic microorganisms and the accumulation of their toxins within the bloodstream. Unlike transient bacteremia, where bacteria are merely transported through blood, septicemia causes profound host immune responses, endothelial damage, disseminated intravascular coagulation, and multi-organ dysfunction, often leading to rapid clinical deterioration.

4. (C) Diffuse petechial hemorrhages and endothelial damage
Acute toxemia occurs when bacterial toxins circulate systemically, targeting endothelial cells lining the blood vessels. This widespread endothelial injury increases vascular permeability and triggers microvascular thrombosis, resulting in characteristic diffuse petechial and ecchymotic hemorrhages across multiple organs and serosal surfaces. This systemic vascular insult is a classic hallmark of toxemic states in acute infectious processes.

5. (A) Both A and R are true and R is the correct explanation of A.
Inhaled viral pathogens commonly utilize the respiratory tract as their portal of entry due to continuous exposure to aerosolized particles. While host defenses like the mucociliary escalator and alveolar macrophages are designed to clear foreign materials, high pathogen loads or specific viral virulence factors can overwhelm these clearance mechanisms, establishing a localized or systemic infection.

6. (D) Phagocytosis by and survival within wandering macrophages
Intracellular bacteria exploit host immune cells for dissemination by surviving inside wandering macrophages after phagocytosis. Instead of being destroyed, these pathogens resist lysosomal degradation, replicate intracellularly, and travel via the lymphatic or vascular systems to distant tissues. This Trojan horse mechanism protects the bacteria from extracellular humoral defenses and facilitates widespread systemic colonization.

NODIA

7. (B) Lymphatic drainage
Following mucosal or tissue invasion, pathogens and antigen-presenting cells are swept from the interstitial spaces into afferent lymphatic vessels. This lymphatic drainage carries the infectious agents directly to regional lymph nodes, which act as filters. Within the lymph nodes, these pathogens encounter immune cells, which either control the infection or fail, leading to nodal pathology.

8. (C) Systemic release of virus following replication in secondary organs
Secondary viremia occurs when a virus replicates extensively in secondary target organs, such as the liver, spleen, or lymph nodes, and is subsequently released back into the bloodstream in high concentrations. This secondary wave is typically much larger than the primary viremia, facilitating widespread dissemination to final target organs and promoting the onset of clinical symptoms.

28. (B) Cell membrane rupture during virion release
In a lytic viral replication cycle, massive replication of viral particles culminates in cell death primarily through cell membrane rupture during virion release. As the viral load peaks, cellular structural integrity is compromised and host metabolic processes are shut down. The physical disruption of the plasma membrane releases newly assembled virions, immediately destroying the host cell.

29. (A) Both A and R are true and R is the correct explanation of A.
Poxviruses are unique among DNA viruses because they replicate exclusively within the cytoplasm of host cells. This cytoplasmic lifecycle is possible because poxviruses encode and package their own DNA-dependent RNA polymerase and other essential enzymes. This machinery allows them to synthesize mRNA independent of host nuclear transcription factors, eliminating the need to enter the cell nucleus.

30. (C) Amyloid deposition
Amyloid deposition is an extracellular pathological accumulation of misfolded fibrillar proteins and is not a direct viral-induced cytopathic effect. In contrast, direct cytopathic effects are structural changes caused by intracellular viral replication, such as apoptosis, cell lysis, or syncytium formation. Amyloidosis instead represents a chronic systemic or localized metabolic disorder linked to chronic inflammation.

31. (D) Splenomegaly with dark, uncoagulated blood
The primary pathognomonic macroscopic lesion of Anthrax in cattle is severe splenomegaly characterized by a very dark, soft, and uncoagulated pulp, often resembling jam. Affected carcasses typically show incomplete rigor mortis, rapid decomposition, and bloody discharges from natural orifices. This failure of blood to clot is due to toxins and vascular damage caused by Bacillus anthracis.

32. (C) Myonecrosis with gas bubbles and hemorrhage
Clostridium chauvoei, the causative agent of Blackleg in ruminants, produces toxins that cause severe localized muscle damage. Histologically, the classic lesion is acute coagulative myonecrosis accompanied by extensive hemorrhage and gas bubbles. These gas bubbles, produced during bacterial fermentation, separate muscle fibers, giving the tissue a porous, dry, and dark reddish-black appearance on gross examination.

NODIA

33. (B) Abscesses and botryomycosis
Staphylococcus infections classically induce purulent inflammation, leading to abscess formation. When chronic, these infections can cause botryomycosis, which is a pyogranulomatous reaction characterized by bacterial colonies surrounded by Splendore-Hoeppli material within granulomatous tissue. This distinguishes staphylococcal disease from other bacterial pathogens that cause non-suppurative or caseating lesions in different organ systems.

34. (D) Microabscesses in the brainstem
Listeriosis in ruminants typically presents as an encephalitic disease caused by Listeria monocytogenes. The primary pathognomonic microscopic finding is the presence of microabscesses, along with focal perivascular cuffing by mononuclear cells, located specifically in the brainstem. These lesions are associated with cranial nerve deficits and circle walking, reflecting localized brainstem damage.

35. (B) II
While Clostridium tetani produces a powerful neurotoxin that causes spastic paralysis by blocking inhibitory neurotransmitter release, it does not cause massive tissue necrosis or gas gangrene. Those necrotic muscle lesions are instead caused by histotoxic Clostridium species such as Clostridium chauvoei, novyi, or perfringens. In contrast, the other statements on anthrax and listeriosis are correct.

36. (C) Edema factor
Edema factor is one of the three protein components of the Anthrax toxin system produced by Bacillus anthracis. When combined with protective antigen, edema factor acts as a calmodulin-dependent adenylate cyclase, elevating intracellular cyclic AMP levels. This disruption of cellular signaling alters water and ion balance, leading to the severe localized edema characteristic of anthrax.

37. (A) Clostridium perfingens type D
Enterotoxemia, or pulpy kidney disease in sheep, is caused by Clostridium perfingens type D. Under favorable dietary conditions, this bacterium proliferates in the gut and secretes a potent protoxin, which is cleaved by trypsin into

94. (C) Narrow, septate hyphae branching at acute angles
In tissue sections, Aspergillus species are histologically identified by the presence of uniform, narrow, septate hyphae that exhibit dichotomous branching at acute angles, approximately 45 degrees. These hyphae readily invade blood vessels, causing local thrombosis, ischemic necrosis, and granulomatous inflammation, which distinguishes them from Mucor species that have broad, unseptate hyphae branching at right angles.

95. (A) P-3, Q-2, R-1, S-4
Microsporum canis causes feline ringworm, producing superficial scaly skin lesions. Candida albicans is the causative agent of thrush, leading to pseudomembranous stomatitis. Aspergillus fumigatus is implicated in bovine mycotic abortion characterized by a thickened placenta. Trichophyton verrucosum is the primary cause of bovine ringworm in calves. These profiles represent specific host-pathogen clinical outcomes.

96. (C) Ringworm
Ringworm, or dermatophytosis, in calves is primarily caused by Trichophyton verrucosum. It is clinically and pathologically characterized by discrete, round, non-pruritic, heavily crusted, and alopecic lesions that occur most commonly on the head, neck, and around the eyes. Histopathology reveals fungal hyphae and arthrospores within the keratinized outer layers and hair follicles.

97. (B) Yeast cells and pseudohyphae
Candida albicans is a dimorphic opportunistic fungus that presents morphologically in tissue lesions as a mixture of budding yeast cells (blastoconidia) and pseudohyphae, often accompanied by true hyphae. This morphological mixture is pathognomonic under microscopic examination. The tissue invasion by these structures triggers local inflammatory infiltration and is a major diagnostic criterion in histopathology.

98. (D) Aspergillosis
Brooder pneumonia is the common name for an acute, severe outbreak of respiratory aspergillosis in young chicks. This condition occurs when neonates inhale massive quantities of Aspergillus fumigatus spores from contaminated litter or incubators. Pathologically, it is defined by the development of multiple, small, yellowish-white granulomatous nodules scattered throughout the lungs and air sacs.

99. (A) Granulomatous
The inflammatory response elicited by tissue invasion of Aspergillus hyphae is predominantly granulomatous to pyogranulomatous. Histopathologically, these lesions feature a central zone of suppurative exudate containing neutrophils and necrotic debris surrounding the invading fungal hyphae, which is enclosed by a prominent outer rim of epithelioid macrophages, multinucleated giant cells, lymphocytes, and fibrovascular tissue.

100. (C) Granulomatous
Systemic mycotic infections, such as those caused by Blastomyces, Histoplasma, or Coccidioides, typically induce a chronic granulomatous or pyogranulomatous inflammatory reaction in host tissues. This protective response is characterized by the accumulation of macrophages, epithelioid cells, and multinucleated giant cells surrounding the fungal elements, attempting to wall off and destroy the persistent organisms.

101. (A) Macrophages
Histoplasmosis is characterized pathologically by the presence of small, round-to-oval yeast cells of Histoplasma capsulatum located primarily within host macrophages. Under light microscopy, these intracellular yeast cells are typically surrounded by a thin, clear, halo-like zone that represents retraction of the cytoplasm rather than a true capsule. This parasitism causes severe histiocytic proliferation.

102. (A) I and II
Dimorphic fungi exist as environmental molds and as yeasts within mammalian host tissues, conforming to statement I. Cryptococcus neoformans possesses a thick mucopolysaccharide capsule, as stated in II. However, Blastomyces dermatitidis replicates by broad-based single budding rather than multiple budding, making statement III incorrect. This single budding is a key distinguishing histopathological feature.

103. (B) Large, broad-based budding yeasts
In tissue sections, Blastomyces dermatitidis is identified as large, spherical, thick-walled yeast cells that reproduce by forming a single bud

Downloaded from www.pdf.tube

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

stimulates osteoclasts, leading to the progressive resorption and atrophy of the nasal turbinate bones. This osteolytic pathology results in facial deformity.

47. (D) F5 (K99) fimbriae
In neonatal calves, enterotoxigenic Escherichia coli utilizes F5, also known as K99, fimbriae to adhere specifically to glycoprotein receptors on the surface of intestinal epithelial cells. This fimbrial adherence is essential for colonization, preventing the bacteria from being cleared by peristalsis. Once attached, the bacteria secrete enterotoxins that cause secretory diarrhea.

48. (B) III
Colisepticemia in calves occurs primarily in those with inadequate colostral transfer, meaning failure of passive transfer of maternal immunoglobulins. Calves with high levels of maternal antibodies are protected against systemic bacterial invasion. Therefore, the statement claiming colisepticemia occurs commonly in calves with adequate colostral immunity is false, while the other statements are correct.

49. (B) Salmonella serovars
The development of 'button ulcers' in the cecum and colon of swine is a pathognomonic lesion of chronic salmonellosis, typically caused by Salmonella Choleraesuis or Salmonella Typhimurium. These distinctive lesions begin as focal necrosis of lymphatic tissue in the intestinal wall, progressing to circular, raised, concentric masses of necrotic debris and fibrin.

50. (D) Angiopathy causing gastric wall and mesentery edema
Edema disease in weaned pigs is caused by Shiga toxin-producing Escherichia coli. The Shiga-like toxin enters circulation and targets endothelial cells of small arteries, causing degenerative angiopathy. This vascular damage increases permeability, leading to the accumulation of edematous fluid in the submucosa of the stomach, mesocolon, forehead, and brain, while avoiding direct mucosal inflammation.

51. (A) Caseating tubercle containing epithelioid and giant cells
The classic histopathological hallmark of bovine tuberculosis, caused by Mycobacterium bovis, is the formation of a caseating tubercle. This chronic granulomatous lesion features a central zone of caseous necrosis, which often undergoes calcification, surrounded by a distinct inflammatory mantle composed of epithelioid macrophages, multinucleated Langhans giant cells, lymphocytes, and an outer fibrous tissue capsule.

52. (C) Ileum, cecum, and colon
Johne's Disease, also known as paratuberculosis, is a chronic granulomatous disease of ruminants that predominantly targets the ileum, cecum, and colon. The causative intracellular pathogen, Mycobacterium avium subsp. paratuberculosis, replicates within macrophages of the lamina propria and submucosa in these specific regions, leading to severe chronic intestinal pathology and clinical wasting.

53. (A) Thickened, corrugated mucosal folds
Grossly, the intestinal mucosa of animals with severe Johne's Disease is characterized by thickened, corrugated mucosal folds that do not flatten when stretched. This pathognomonic appearance is caused by the massive infiltration and accumulation of epithelioid macrophages and giant cells in the lamina propria and submucosa, which compromises the nutrient absorptive capacity of the intestine.

54. (D) Pyogranulomatous osteomyelitis of the jaw
Actinomycosis, commonly referred to as lumpy jaw, is caused by the Gram-positive bacterium Actinomyces bovis. This opportunistic pathogen enters through oral mucosal wounds and invades the mandible or maxilla, initiating a chronic pyogranulomatous osteomyelitis. This bone tissue reaction leads to severe osteolysis, periosteal bone deposition, and fistulous tracts discharging pus containing characteristic sulfur granules.

55. (A) P-4, Q-3, R-2, S-1
Mycobacterium bovis causes caseating lung tubercles. Mycobacterium paratuberculosis causes chronic corrugated intestinal changes in Johne's Disease. Actinomyces bovis targets bovine jaw bones, leading to pyogranulomatous osteomyelitis. Actinobacillus lignieresii causes granulomatous glossitis, known as wooden tongue. These distinct tissue reactions reflect differences in target tissue tropism and immune interactions of each specific pathogen.

NODIA

75. (B) Atrophy and blunting of the apical villi
Rotaviruses specifically target and replicate within mature, absorptive enterocytes at the tips and sides of the intestinal villi. The destruction and loss of these cells lead to marked villous atrophy, shortening, and blunting of the apical villi, while sparing the crypt cells. This compromise of the mucosal surface area results in malabsorptive diarrhea.

76. (A) I, II and III
Foot-and-Mouth Disease, Peste des Petits Ruminants, and Rotaviral infections conform to statements I, II, and III respectively. However, Classical Swine Fever does not cause severe splenomegaly without splenic infarcts; instead, the presence of marginal splenic infarction is a highly characteristic and pathognomonic lesion of Classical Swine Fever, making statement IV incorrect.

77. (A) Spongiosis and ballooning degeneration
Histologically, Foot-and-Mouth Disease virus replication within stratified squamous epithelial cells of the oral mucosa induces intercellular edema (spongiosis) and intracellular edema, known as ballooning degeneration. These cytopathic changes cause affected epithelial cells to swell, lose their cell-to-cell adhesions, and undergo lysis, creating the microvesicles that eventually coalesce into macroscopically visible vesicles.

78. (C) Zebra striping in the colon and rectum
In Peste des Petits Ruminants, mucosal congestion and hemorrhages in the large intestine are distributed along the longitudinal mucosal folds of the colon and rectum. This produces a highly characteristic gross lesion referred to as 'zebra striping'. This lesion reflects severe local vascular damage and is a primary diagnostic indicator of morbillivirus pathology in small ruminants.

79. (D) Perivascular cuffing and glial nodules
The non-suppurative encephalitis observed in pigs with Classical Swine Fever is histopathologically characterized by prominent perivascular cuffing by lymphocytes and macrophages, alongside focal microglial proliferation forming glial nodules. These inflammatory changes reflect the neurotropic nature of the systemic virus, representing a typical viral encephalitic reaction rather than a purulent or necrotizing bacterial response.

80. (C) Eosinophilic intracytoplasmic Negri bodies
The pathognomonic histological lesion for Rabies in the central nervous system is the presence of eosinophilic, round-to-oval, intracytoplasmic inclusion bodies known as Negri bodies. These structures consist of viral ribonucleoproteins and are found in infected neurons. While non-suppurative meningoencephalitis and perivascular cuffing also occur, Negri bodies provide definitive histopathological confirmation of rabies.

81. (B) Skeletal muscle
Canine Distemper virus is a pantropic morbillivirus that exhibits strong tropism for epithelial tissues, lymphoid tissues, and the central nervous system. It infects epithelial cells of the respiratory, gastrointestinal, and urogenital tracts, as well as cerebral glial cells and neurons. However, the virus does not typically target or cause pathology in skeletal muscle tissue.

82. (A) II, I, III, IV
The pathogenesis of Rabies begins when the virus is introduced via a bite wound from an infected animal. It initially replicates within local muscle tissue at the inoculation site before entering neuromuscular junctions. From there, the virus ascends via retrograde axoplasmic flow along peripheral nerves to the central nervous system, followed by centrifugal spread to salivary glands.

83. (A) Hemorrhagic tracheitis and pneumonia
Newcastle Disease virus, particularly velogenic strains, causes severe, acute respiratory system lesions in poultry. Pathological findings typically include severe hemorrhagic tracheitis, characterized by petechial and ecchymotic hemorrhages in the tracheal mucosa, along with interstitial pneumonia and airsacculitis. These extensive respiratory lesions disrupt oxygen exchange, contributing significantly to the high mortality rates in affected flocks.

84. (C) Demyelination of white matter tracts
A characteristic histopathological finding in Canine Distemper encephalitis is demyelination of white matter tracts, especially in the cerebellum and around the fourth ventricle. This demyelination occurs in both acute and chronic phases of the disease. It is caused by direct viral

Click Here to Buy book on Amazon

65. (A) Both A and R are true and R is the correct explanation of A.
Parvovirus replication is strictly dependent on host cells that are undergoing active cell division, specifically in the S-phase of the cell cycle. The intestinal crypt epithelium is characterized by a high mitotic index, making it highly susceptible. Destruction of these dividing crypt progenitor cells halts mucosal regeneration, resulting in severe villous atrophy and necrotizing enteritis.

66. (D) Wart-like, scabby skin lesions
The cutaneous, or dry, form of fowl pox is pathologically defined by the development of discrete, proliferative, wart-like nodules on non-feathered regions of the skin, such as the comb, wattles, and eyelids. These epithelial lesions quickly progress from vesicles to thick, dark brown scabs, reflecting hyperplastic epidermal growth driven by poxvirus replication within keratinocytes.

67. (A) Cowdry Type A
Canine Adenovirus type 1 replication produces prominent, large, eosinophilic to amphophilic intranuclear inclusion bodies, classified as Cowdry Type A. These inclusion bodies are found within infected hepatocytes, biliary epithelial cells, and vascular endothelial cells. Under light microscopy, they occupy a large portion of the nucleus and are surrounded by a distinct clear halo with marginated chromatin.

68. (C) Thickening and loss of striation in peripheral nerves
A classical macroscopic feature of Marek's Disease is the bilateral or unilateral thickening and loss of normal cross-striations in peripheral nerves, particularly the sciatic, vagus, and brachial nerves. This nerve swelling, caused by the infiltration of pleomorphic T-lymphocytes, gives the affected nerves a dull, yellowish-gray, and edematous appearance, leading to paralysis.

69. (A) Oropharyngeal lymphoid tissue
Following transmission via the oronasal route, Canine Parvovirus initially replicates within active lymphoid tissues, specifically the tonsils and oropharyngeal lymph nodes. After this local replication, the virus enters the bloodstream, causing a systemic viremia that distributes the virus to secondary targets, including the bone marrow, lymphoid organs, and intestinal crypt epithelial cells.

70. (D) Vesicles on oral mucosa and feet
Foot-and-Mouth Disease is a highly contagious aphthovirus infection of cloven-hoofed animals. It is pathologically characterized by the formation of vesicles (aphthae) on the oral mucosa, dental pad, tongue, coronary band, and interdigital skin of the feet. These fluid-filled vesicles rapidly rupture, leaving painful erosions that lead to salivation, lameness, and decreased feed intake.

71. (B) Respiratory and digestive systems
Peste des Petits Ruminants is an acute, highly contagious viral disease of sheep and goats caused by a morbillivirus. It primarily targets the respiratory and digestive systems, leading to severe pathological changes. These lesions manifest as necrotizing and erosive stomatitis, severe enteritis with diarrhea, and an interstitial to exudative bronchopneumonia, which together cause high mortality.

72. (C) Myocardial degeneration and necrosis
In young calves, Foot-and-Mouth Disease virus exhibits a strong cardiotropic tendency, causing high mortality without the typical vesicular skin lesions seen in adult animals. Pathologically, this manifests as severe acute myocardial degeneration and coagulative necrosis, which appears grossly as greyish-white streaks in the myocardium, historically termed "tiger heart," leading to heart failure.

NODIA

73. (D) Button ulcers in the cecum and colon
Classical Swine Fever, caused by a Pestivirus, classically induces concentric necrotizing mucosal lesions, known as button ulcers, in the cecum and colon. These lesions occur due to viral-induced ischemic necrosis from localized vascular damage and thrombosis of submucosal blood vessels. These circular ulcers are often complicated by secondary opportunist bacterial pathogens, such as Salmonella.

74. (A) Endothelial cells
Classical Swine Fever virus exhibits a strong tropism for vascular endothelial cells and lymphoid tissues. Direct viral replication inside these endothelial cells causes endothelial degeneration, swelling, and desquamation, leading to widespread vascular damage and microvascular thrombosis. This generalized angiopathy manifests as petechial and ecchymotic hemorrhages in multiple organs, including the kidneys, bladder, and larynx.

56. (C) Bacterial colonies with Splendore-Hoeppli material
The 'sulfur granules' observed in pus from Actinomycosis lesions are macroscopic colonies of Actinomyces bovis surrounded by an eosinophilic, club-like border of Splendore-Hoeppli material. This material consists of precipitated host immunoglobulins, complement proteins, and cellular debris. The presence of these mineralized or protein-coated colonies is highly characteristic of chronic pyogranulomatous infections in livestock.

57. (D) Cord factor
Cord factor, chemically known as trehalose 6,6'-dimycolate, is an important glycolipid in the Mycobacterial cell wall. This molecule is responsible for the parallel alignment of bacteria in culture and plays a major role in virulence by inhibiting phagosome-lysosome fusion within macrophages. Additionally, cord factor induces the formation of granulomatous inflammation and caseous necrosis.

58. (A) Macrophages
Epithelioid cells, the primary constituents of granulomatous lesions in chronic infections like tuberculosis, are specialized activated macrophages. Under the influence of interferon-gamma and other cytokines, these macrophages undergo morphological changes, developing abundant cytoplasm and closely apposed cell borders that resemble epithelial cells. These modified cells are optimized for extracellular secretion rather than phagocytosis.

59. (B) Diarrhea and wasting
In Johne's Disease, the massive infiltration of epithelioid macrophages in the lamina propria and submucosa of the intestine severely impairs mucosal function. This chronic inflammation leads to malabsorption, causing persistent, intractable diarrhea and progressive wasting (cachexia) in affected animals. This occurs despite a normal or increased appetite, as nutrients cannot be absorbed.

60. (B) Ballooning degeneration with intracytoplasmic inclusions
Poxviruses induce a classic histopathological reaction in epidermal cells characterized by ballooning degeneration and the presence of eosinophilic intracytoplasmic inclusion bodies, known as Bollinger or Guarnieri bodies. Ballooning degeneration represents severe intracellular edema where infected epithelial cells swell and lose their intercellular attachments. This characteristic cytopathic change is central to the formation of cutaneous pox vesicles.

61. (A) Hemorrhagic tracheitis with intranuclear inclusions
Infectious Laryngotracheitis is a severe herpesviral disease of poultry. The pathognomonic lesion in the trachea is an acute hemorrhagic to diphtheritic tracheitis. Histologically, infected tracheal epithelial cells exhibit syncytial cell formation accompanied by prominent Cowdry type A intranuclear inclusion bodies. These inclusions are most readily observed in the early stages of the respiratory infection.

62. (C) Neoplastic T-lymphocyte infiltration in nerves and organs
Marek's Disease is a lymphoproliferative disease of chickens caused by a herpesvirus. It is pathologically characterized by the neoplastic transformation and infiltration of T-lymphocytes into peripheral nerves and visceral organs. This lymphoid infiltration causes nerve enlargement, leading to paralysis, as well as macroscopic tumor formation in the liver, spleen, kidneys, heart, and skin tissues.

63. (D) Liver and vascular endothelium
Infectious Canine Hepatitis, caused by Canine Adenovirus type 1, exhibits strong tropism for hepatocytes and vascular endothelial cells. This replication results in severe hepatic necrosis and generalized vascular injury. Pathological hallmarks include centrilobular hepatic necrosis, gallbladder wall edema, petechial hemorrhages across multiple organs, and the presence of large, basophilic intranuclear inclusion bodies within hepatocytes and endothelial cells.

64. (C) Necrotizing enteritis and myocarditis
In young puppies, Canine Parvovirus targets highly mitotically active cells because it requires host cell DNA replication machinery. In puppies under a few weeks of age, this leads to replication in developing cardiac myocytes, causing acute myocarditis. In slightly older puppies, the virus infects rapidly dividing intestinal crypt cells, resulting in severe necrotizing enteritis and villous atrophy.

Downloaded from www.pdf.tube

damage to myelin-producing oligodendrocytes as well as subsequent immune-mediated responses against myelin antigens.

85. (A) Hemorrhages in the proventriculus and gut lymphoid patches
Viscerotropic velogenic Newcastle Disease is characterized by severe hemorrhages and necrosis in the visceral organs of poultry. Pathognomonic gross lesions include prominent petechial and ecchymotic hemorrhages in the mucosa of the proventriculus, particularly at the tips of the glands, along with severe hemorrhagic necrosis of the cecal tonsils and intestinal lymphoid patches.

86. (D) Hippocampus (carnivores) and Purkinje cells (herbivores)
When diagnosing Rabies, the neuroanatomical distribution of Negri bodies varies across species. In carnivores, such as dogs and cats, these eosinophilic intracytoplasmic inclusions are most consistently found within the pyramidal cells of the hippocampus. In contrast, in herbivores, such as cattle and sheep, they are most reliably located within the Purkinje cells of the cerebellum.

87. (B) Canine Distemper
Canine Distemper virus infection often manifests pathologically as hyperkeratosis of the footpads and planum nasale, a condition clinically termed hard pad disease. This lesion is caused by viral replication within epithelial keratinocytes, which disrupts normal epidermal maturation and leads to excessive accumulation of keratin layers. This hyperkeratosis is a key diagnostic indicator in dogs.

88. (B) III
Newcastle Disease is not restricted solely to the nervous system in birds; indeed, velogenic and mesogenic pathotypes cause extensive systemic lesions, including severe hemorrhagic enteritis, proventriculitis, and respiratory tract pathology. The other statements correctly describe the retrograde transport of Rabies virus, the immunosuppressive and demyating mechanisms of Canine Distemper virus, and the composition of Negri bodies.

89. (D) Intranuclear and intracytoplasmic
Canine Distemper virus is unique among paramyxoviruses because it produces both intranuclear and intracytoplasmic inclusion bodies within infected host cells. These eosinophilic inclusion bodies are most commonly observed in epithelial cells of the respiratory tract, urinary bladder, gastrointestinal mucosa, and skin, as well as in neurons and glial cells, serving as a critical histopathological diagnostic feature.

90. (A) Keratinized structures like hair and nails
Dermatophytosis, commonly known as ringworm, is a superficial fungal infection caused by dermatophytes that possess keratinolytic enzymes. These fungi specifically colonize and invade non-living keratinized structures of the epidermis, including the stratum corneum, hair shafts, and nails or claws. They do not invade living epidermal cells, the underlying dermis, or deeper subcutaneous tissues.

NODIA

91. (B) Hyperkeratosis and folliculitis with hyphae
Histopathologically, dermatophytosis is characterized by orthokeratotic or parakeratotic hyperkeratosis, accompanied by luminal folliculitis. The fungal hyphae and arthroconidia can be observed invading the hair follicles and shafts, often highlighted with special stains. This fungal presence triggers local inflammatory infiltration of neutrophils and mononuclear cells, leading to follicular rupture and localized dermatitis.

92. (B) Thickened, grayish-white crop pseudomembrane
Candidiasis, commonly called thrush or sour crop in birds, is an opportunistic infection of the upper digestive tract. It is pathologically characterized by the formation of a thickened, grayish-white, circular, or confluent pseudomembrane that tightly adheres to the mucosal lining of the crop and esophagus, resembling a Turkish towel on gross examination.

93. (A) Caseous nodules in the lungs and air sacs
Aspergillosis in birds is primarily a respiratory infection caused by Aspergillus fumigatus. Pathologically, it manifests as discrete, yellowish-white, caseous nodules within the parenchyma of the lungs and on the membranes of the air sacs. In severe cases, these granulomatous lesions can coalesce to form large plaques covered with green-gray fungal matting during active sporulation.

active epsilon toxin. This toxin enters systemic circulation, causing vascular endothelial damage, severe edema in multiple organs, and encephalomalacia.

38. (D) Botryomycosis
Botryomycosis is a chronic pyogranulomatous bacterial infection characterized microscopically by the presence of Splendore-Hoeppli material surrounding aggregates of Gram-positive cocci, typically Staphylococcus species. This eosinophilic, club-like matrix represents antigen-antibody complexes and host proteins deposited around the bacteria. This morphology distinguishes botryomycosis from actinomycosis, which involves filamentous bacteria, and actinobacillosis, which involves Gram-negative bacilli.

39. (B) Listeria monocytogenes
Listeria monocytogenes is a facultative intracellular bacterium that has evolved a specialized mechanism to spread between host cells. After escaping from the phagosome, it polymerizes host cell actin to form tail-like structures called actin tails. These tails propel the bacterium through the cytoplasm and directly into neighboring cells, bypassing the extracellular environment and humoral defenses.

40. (C) Incomplete or absent
In animals that die suddenly from Anthrax, rigor mortis is typically incomplete or completely absent. The high concentration of Bacillus anthracis toxins in the blood causes severe damage to blood vessels and rapid degradation of tissues, which prevents the normal muscular contractions and cellular changes necessary to establish rigor mortis. This is a key diagnostic indicator.

41. (D) Enterotoxin-mediated fluid hypersecretion
Enterotoxigenic Escherichia coli causes colibacillosis in young calves, presenting as white scours. The primary pathological mechanism relies on enterotoxins that stimulate cyclic nucleotide pathways in enterocytes without causing mucosal structural damage. This activation leads to hypersecretion of fluids and electrolytes into the lumen, resulting in profuse, watery diarrhea and rapid dehydration while keeping the microvilli intact.

42. (A) Ingestion → Invasion of enterocytes and M cells → Multiplication in macrophages → Acute inflammatory response
Salmonella-induced enterocolitis begins with the oral ingestion of the bacteria, which travel to the distal ileum and colon. The bacteria then invade enterocytes and specialized M cells. Following entry, they are engulfed by macrophages, where they survive and multiply. This intracellular survival and replication trigger a massive recruit of neutrophils, causing an acute inflammatory response.

43. (B) Mannheimia haemolytica
Mannheimia haemolytica is the principal pathogen responsible for shipping fever in cattle. The classic gross lesion is severe fibrinous pleuropneumonia showing a marbled appearance. This marbling of the lungs is caused by distension of the interlobular septa by fibrinous exudate, edema, and lymphatic congestion, which stands out against the dark, consolidated hepatized pulmonary lobules.

44. (B) Necrotizing placentitis with leathery intercotyledonary areas
Brucella abortus targets the pregnant bovine uterus because of the presence of erythritol, which stimulates its growth. The hallmark lesion is a severe necrotizing placentitis where the cotyledons appear necrotic and yellowish-gray, while the intercotyledonary areas become thickened, opaque, and leathery. This compromise of the placenta disrupts fetal support, resulting in abortion.

45. (A) Hepatic paratyphoid nodules and colonic button ulcers
In swine, infection with Salmonella enterica serovar Choleraesuis classically leads to systemic salmonellosis. Pathological findings include focal necrosis in the liver known as paratyphoid nodules, which are microgranulomas composed of macrophages and necrotic hepatocytes. Additionally, chronic infection causes localized mucosal necrosis in the cecum and colon, manifesting as distinct button ulcers.

46. (C) Atrophic Rhinitis
Atrophic rhinitis in swine is caused by co-infection of Bordetella bronchiseptica and toxigenic strains of Pasteurella multocida type D. The dermonecrotic toxin produced by Pasteurella multocida type D inhibits osteoblasts and

Click Here to Buy book on Flipkart

attached to the mother cell by a characteristic broad base. These yeast cells are surrounded by a prominent double-contoured cell wall. They typically elicit a pyogranulomatous inflammatory response in affected organs, particularly the lungs and skin.

104. (C) The thick polysaccharide yeast capsule
The gelatinous appearance of tissue lesions in feline cryptococcosis is due to the accumulation of numerous Cryptococcus neoformans yeast cells, each surrounded by a thick, mucoid, extracellular polysaccharide capsule. This voluminous capsule prevents close contact between yeasts and host inflammatory cells, resulting in a minimal cellular response and giving the gross lesions their distinct translucent, mucoid quality.

105. (D) Mucicarmine stain
The thick mucopolysaccharide capsule of Cryptococcus neoformans can be visualized in tissue sections using mucicarmine stain, which stains the capsule a brilliant red or pink against a yellow or blue background. This selective staining is highly diagnostic, enabling pathologists to easily distinguish Cryptococcus neoformans from other non-encapsulated yeasts like Histoplasma and Blastomyces in histopathology.

106. (B) Thickened, corrugated mucosa with massive macrophage infiltration
Histoplasma capsulatum typically infects the gastrointestinal tract of dogs, where it replicates inside macrophages. This causes a severe granulomatous enteritis characterized by a thick, corrugated mucosal layer. The cellular response is dominated by massive macrophage infiltration containing intracellular yeast. These changes compromise nutrient absorption, resulting in progressive wasting and chronic diarrhea.

107. (A) Ulcerated, draining pyogranulomatous skin nodules
Cutaneous blastomycosis in veterinary patients represents a common dissemination of the systemic fungal infection. The organism causes a robust pyogranulomatous inflammatory response within the dermis and subcutis. This reaction produces raised, ulcerated, and draining skin nodules. Exudate from these skin lesions typically contains numerous broad-based budding yeast cells mixed with neutrophils and macrophages.

108. (C) A yeast
Blastomyces dermatitidis is a thermally dimorphic fungus, existing in different structural forms depending on environmental temperature. In natural soil environments at cooler ambient temperatures, the organism grows as a saprophytic mold. Once inhaled or introduced into a mammalian host at body temperature, the fungus shifts morphologically to replicate as a thick-walled pathogenic yeast.

109. (D) Ascaris suum within the small intestine
Large parasitic nematodes residing in narrow luminal spaces can physically block normal transit of digested material. High burdens of these mature roundworms in the small intestine of swine can physically occlude the lumen. This mechanical obstruction leads to severe impaction, local pressure necrosis, and can cause life-threatening intestinal rupture in affected animals.

110. (C) Echinococcus granulosus hydatid cysts
Metacestode stages of specific tapeworms form slow-growing, fluid-filled cysts in various host tissues. As these structures expand in size, they exert constant physical pressure on the surrounding parenchymal cells. This chronic pressure results in localized compressive atrophy of the affected organ, such as liver or lung tissue, compromising normal physiological function.

111. (B) Migrating Fasciola hepatica or Ascaris suum larvae
Immature stages of certain helminths must physically traverse host tissues to reach their final site of maturation. During this migration, their physical movement causes direct trauma, microvascular injury, and enzymatic destruction. This leads to distinct linear tracts of parenchymal necrosis and hemorrhage, which eventually resolve by forming dense lines of fibrous tissue.

112. (D) Adult intestinal cestodes such as Moniezia
Adult tapeworms residing within the host gastrointestinal lumen do not feed on host blood or tissues. Instead, they absorb dissolved nutrients directly across their specialized tegument, competing with the host for essential dietary components. Large numbers of these parasites can lead to metabolic deficiencies, unthriftiness, and poor growth rates in young grazing ruminants.

Downloaded from www.pdf.tube

113. (A) Eosinophils
Parasitic antigens often stimulate a helper T-cell type two response, which promotes the synthesis of interleukin-5. This specific cytokine is critical for the recruitment, proliferation, and activation of eosinophils in tissues. Consequently, parasitic infections and associated hypersensitivity reactions are classically characterized by prominent eosinophilic infiltrates aimed at neutralizing the invading organisms.

114. (C) Focal fibrosis from migrating Ascaris suum larvae
When swine ingest infective eggs, the larval stages hatch and migrate through the intestinal wall, reaching the liver via the portal vein. During their migration through hepatic parenchyma, these larvae cause tissue destruction, hemorrhage, and inflammation. The host resolves these focal injuries by depositing fibrous connective tissue, visible as white scars known as milk spots.

115. (A) Both A and R are true and R is the correct explanation of A.
In grazing sheep, the blood-feeding abomasal nematode consumes significant quantities of host blood daily. This voracious feeding activity leads directly to blood loss, resulting in regenerative anemia and depletion of plasma proteins. The clinical manifestations of severe anemia and submandibular edema are direct physiological consequences of the parasite feeding on the abomasal mucosa.

116. (D) Endothelial injury leading to arteritis, thrombosis, and infarction
Migrating larvae of specific strongyles penetrate the intestinal wall of horses and migrate along the intima of the cranial mesenteric artery. This migration causes mechanical damage to the vascular endothelium, promoting localized inflammatory arteritis. This vascular damage triggers thrombus formation, which can occlude blood flow, causing bowel infarction, colic, and potential intestinal necrosis.

117. (B) Hydatid cyst
Metacestode stages of specific tapeworms form large, fluid-filled cysts in various organs. The fluid contained within these structures is highly antigenic to the host. If a cyst ruptures spontaneously or during surgical removal, the sudden systemic release of this fluid triggers a massive, immediate hypersensitivity reaction, leading to life-threatening anaphylactic shock.

118. (C) Wall off and destroy parasite eggs or trapped larvae
Eosinophils are recruited to sites of parasitic infection by chemotactic factors. When parasitic eggs or larval stages are too large to be phagocytosed, eosinophils cluster around them, releasing cytotoxic granule proteins to damage the parasite. Over time, macrophages and fibroblasts surround this focus, forming a structured granuloma that isolates the foreign material.

119. (D) Severe anemia, abomasal mucosal damage, and peripheral edema
Ingestion of infective larvae of this abomasal nematode leads to mucosal colonization and blood feeding. The physical disruption of the gastric mucosa by mature worms causes protein-losing enteropathy and hemorrhage. Clinically, this results in severe regenerative anemia, mucosal injury, hypoproteinemia, and the accumulation of fluid in the submandibular space, commonly called bottle jaw.

NODIA

120. (A) Bronchi and bronchioles, causing catarrhal bronchitis
Dictyocaulus viviparus larvae migrate from the intestinal tract via the lymphatic system to the lungs of cattle. Upon reaching the pulmonary tissue, they mature within the lumina of the bronchi and bronchioles. Their presence and movement cause mechanical irritation and inflammation, leading to a severe catarrhal bronchitis characterized by mucus accumulation, coughing, and dyspnea.

121. (B) The nodular worm Oesophagostomum columbianum
In many sheep, ingestion of infective larvae of this nematode leads to penetration of the intestinal wall, particularly in the colon. The host immune response walls off the larvae in the submucosa, forming small, localized caseous or calcified nodules. When these are numerous, they give the intestinal serosa a characteristic bumpy, pimply appearance.

122. (B) Mechanical impaction or acute intestinal rupture
Parascaris equorum is a very large roundworm that infects foals. When these animals harbor

high parasite burdens, the sheer volume of adult worms can completely fill the intestinal lumen. This causes physical blockage and severe impaction. If the pressure increases or if the worms move actively, they can cause acute rupture of the bowel.

123. (A) Dictyocaulus filaria
Dictyocaulus filaria is the primary lungworm species affecting sheep and goats. The adult nematodes inhabit the bronchi and bronchioles of the lower respiratory tract. Their presence causes chronic mechanical irritation, leading to a catarrhal inflammatory response. This results in mucous accumulation, localized lung collapse, coughing, and reduced respiratory efficiency in affected small ruminants.

124. (C) Abomasal gland hyperplasia from emerging Ostertagia larvae
Ingestion of Ostertagia ostertagi larvae leads to their entry into the gastric glands of the cattle abomasum. As the larvae develop and grow, they cause compression and hyperplasia of the surrounding glandular epithelium. The emergence of these larvae disrupts the mucosal structure, resulting in a thick, rough mucosa with a characteristic leather-like texture.

125. (C) I, II and III
Haemonchus contortus is a blood-feeding nematode that causes severe anemia. Dictyocaulus viviparus is the bovine lungworm that causes bronchitis. Oesophagostomum species cause nodule formation in the intestines of sheep and pigs. However, the milk spots observed on the pig liver are caused by migrating larvae of Ascaris suum, not by the adult intestinal worms.

126. (C) The brain, eyes, and visceral organs
Visceral larva migrans occurs when accidental hosts ingest infective eggs of Toxocara canis. The larvae hatch, penetrate the gut, and migrate widely through various tissues. Because they cannot mature, they become trapped. The host immune response forms eosinophilic granulomas around these larvae in organs such as the brain, eyes, and visceral tissues.

127. (B) Trichuris vulpis
Trichuris vulpis is the canine whipworm, named for its characteristic thin anterior end and thick posterior end. This nematode targets the cecum and proximal colon, where it burrows its slender anterior end deep into the mucosal epithelium. This structural attachment causes mechanical irritation, mucosal damage, and localized typhlitis or colitis in affected dogs.

128. (D) Intestinal mucosal damage and acute blood loss anemia
Hookworms attach to the mucosa of the small intestine using their specialized buccal capsule, shearing the host tissue. They secrete anticoagulant proteins and actively ingest blood from the damaged vessels. The constant movement of these parasites to new feeding sites causes persistent focal hemorrhages, resulting in severe hypoalbuminemia and acute blood loss anemia.

129. (A) Traumatic hepatitis, hemorrhagic tracts, and sudden death
Acute fasciolosis is caused by the migration of large numbers of immature flukes through the liver parenchyma of sheep. Their physical movement and feeding destroy hepatocytes, causing traumatic hepatitis, extensive parenchymal necrosis, and internal bleeding. If severe enough, this widespread parenchymal destruction and blood loss can lead to sudden death in affected grazing sheep.

130. (C) Biliary hyperplasia and periductal fibrosis ('pipe-stem' liver)
Chronic fasciolosis in cattle occurs when adult flukes establish residency in the bile ducts. Their physical presence, along with spines on their tegument and toxic excretions, causes chronic irritation to the biliary tract. This results in extensive biliary epithelial hyperplasia, marked periductal fibrosis, and calcification, leading to a classic pipe-stem liver appearance.

131. (A) Echinococcus granulosus
Echinococcus granulosus is a tiny tapeworm that resides in the small intestine of dogs. The intermediate stages develop in herbivores or humans after ingesting eggs. These larval forms develop into slow-growing, fluid-filled structures called hydatid cysts. These cysts contain a thick germinal layer that buds off multiple infective protoscoleces, causing compressive tissue damage.

Click Here to Buy book on Amazon

188. (C) Severe bilateral conjunctivitis and rhinitis
Chlamydia felis is a highly infectious pathogen of cats. It has a specific tissue tropism for the mucosal membranes of the ocular and upper respiratory systems. In affected felines, the infection causes severe, acute bilateral conjunctivitis characterized by chemosis, blepharospasm, and serous to mucopurulent ocular discharge, often accompanied by mild rhinitis.

189. (D) Conformational change of a normal cellular protein
The pathogenesis of transmissible spongiform encephalopathies is centered on a post-translational event. It involves the conformational conversion of the normal, host-encoded cellular prion protein into a pathogenic, misfolded isoform. This abnormal protein serves as a template, inducing other normal prion molecules to misfold, leading to accumulation, neurotoxicity, and progressive brain damage.

190. (B) II and III
The pathogenic prion isoform possesses unique biochemical properties that differentiate it from the normal cellular protein. Structurally, it is enriched with beta-pleated sheets rather than alpha-helices. This conformational change makes the protein highly resistant to standard enzymatic and protease degradation, facilitating its accumulation as insoluble fibrils and amyloid plaques in the brain.

191. (B) Alpha-helices
The normal, host-encoded cellular prion protein is a cell-surface glycoprotein expressed predominantly on the membranes of neurons. Under normal physiological conditions, the tertiary structure of this protein is highly soluble and is composed predominantly of alpha-helices with very few beta-sheets. This structure allows the protein to be easily degraded by normal cellular proteases.

192. (D) Lacks any nucleic acid genome
Transmissible spongiform encephalopathies are etiologically unique among infectious diseases. Standard infectious agents, such as viruses, bacteria, and fungi, rely on DNA or RNA genomes to replicate. Conversely, the infectious prion agent is purely proteinaceous, lacking any nucleic acid genome, and transmits infection by template-directed refolding of the host normal cellular protein.

193. (A) Ingestion of contaminated meat-and-bone meal
Bovine Spongiform Encephalopathy was identified as an epidemic disease of cattle in the United Kingdom during the late twentieth century. Epidemiological investigations demonstrated that the primary mode of transmission was oral. Cattle became infected by ingesting concentrates containing meat-and-bone meal that was contaminated with scrapie-like prions or recycling of bovine prions.

194. (C) Proteosomal and lysosomal degradation
The normal cellular prion protein undergoes regular endocytosis and is completely degraded by host lysosomal enzymes and proteasomes. However, the misfolded pathogenic isoform is highly resistant to enzymatic cleavage. This conformational stability prevents normal lysosomal and proteasomal degradation, leading to its accumulation within the endolysosomal system, resulting in cellular toxicity.

195. (A) III, II, I, IV
The pathogenesis of orally acquired prion diseases follows a defined anatomical pathway. After ingestion, the resistant prions survive digestion and are absorbed in the gut, accumulating within Peyer's patches. They then utilize the enteric nervous system to migrate retrogradely along autonomic nerves to the brainstem, finally causing widespread neuronal vacuolation.

196. (D) At the cell membrane or endocytic vesicles
The conversion of the normal cellular prion protein to the pathogenic misfolded isoform is a critical molecular event. This conformational change does not occur inside the cell nucleus or mitochondria. Instead, current research indicates that the misfolding process is initiated primarily at the cell membrane or within early endocytic vesicles during recycling.

197. (B) Neuronal vacuolation and neuropil spongiform change
Histopathological diagnosis of transmissible spongiform encephalopathies is based on identifying characteristic changes in brain sections. The hallmark lesion is characterized by the presence of multiple, clear vacuoles within the perikaryon of neurons. Additionally, widespread vacuolation is observed in the neuropil, giving the gray matter of the brain a classic spongiform appearance.

NODIA

of infected red cells in brain capillaries, causing severe microvascular congestion, tissue hypoxia, and cerebral dysfunction.

142. (C) Lymphocytes
Theileria parva is the tick-borne protozoan responsible for East Coast Fever in cattle. Upon entry into the host system, the sporozoites specifically target and invade mononuclear cells, primarily T-lymphocytes and B-lymphocytes. Inside these host immune cells, the parasite replicates as a schizont, inducing clonal expansion and uncontrolled proliferation of the infected lymphoid cells.

143. (B) Koch's blue bodies (schizonts)
When lymphocytes are infected with Theileria, the sporozoites develop into multinucleated intracellular structures. These active schizonts can be identified using Giemsa-stained lymph node or spleen biopsies. Macroscopically, they appear as small, distinct blue-purple inclusions within the cytoplasm of lymphocytes. In veterinary diagnostics, these pathognomonic diagnostic structures are commonly referred to as Koch's blue bodies.

144. (C) III
Babesia species replicate within erythrocytes, leading directly to acute intravascular hemolysis and icterus. Theileria annulata causes an acute lymphoproliferative phase followed by profound lymphoid depletion. However, animal trypanosomiasis is characterized primarily by persistent extravascular hemolytic processes and bone marrow suppression. This makes the statement concerning consistent acute intravascular hemolysis in trypanosomiasis incorrect.

145. (A) Undergo antigenic variation of its surface glycoproteins
Trypanosomes live extracellularly within the bloodstream of host animals. Their primary survival mechanism is their ability to continually alter their variable surface glycoproteins. This process of antigenic variation prevents the host immune system from clearing the infection, leading to cyclical waves of parasitemia, chronic vascular inflammation, tissue damage, and progressive cachexia.

146. (C) Markedly enlarged, dark, and soft (splenomegaly)
In acute cases of babesiosis, the massive intravascular hemolysis of host erythrocytes triggers a rapid response in the mononuclear phagocyte system. The spleen becomes heavily congested due to the active phagocytosis of damaged red blood cells and free hemoglobin. This massive congestion results in a greatly enlarged, dark, and soft spleen.

147. (A) Extravascular hemolytic and dyshemopoietic
Trypanosomiasis causes anemia through multiple mechanisms, but intravascular lysis is minimal. Instead, circulating red blood cells are opsonized and subsequently removed by macrophages in the spleen and liver. Concurrently, chronic inflammation leads to impaired iron utilization and bone marrow suppression, resulting in a combination of extravascular hemolysis and dyshemopoietic anemia in the host.

148. (D) Massive proliferation of parasitized lymphocytes
In the early phase of East Coast Fever, sporozoites invade host lymphocytes. The parasite replicates as a schizont and actively drives the transformation of host cells. This interaction triggers uncontrolled, clonal division of the infected lymphocytes, similar to a neoplastic process. This massive proliferation occurs throughout lymphoid tissues, causing generalized lymphadenopathy.

149. (B) Ceca
Eimeria species demonstrate high tissue specificity within the avian gastrointestinal tract. Eimeria tenella is a highly pathogenic species that localizes in the ceca of poultry. The parasite invades and replicates within the cecal epithelial cells and lamina propria, causing severe necrosis, mucosal thickening, hemorrhage, and classic bloody feces in young chickens.

150. (C) Rupture of enterocytes during release of merozoites
Intestinal coccidiosis pathology is driven by the intracellular replication of the parasite. During the schizogony phase, numerous merozoites form inside host enterocytes. When these merozoites mature, they exit the cell, causing the physical rupture of the host enterocyte. Widespread host cell lysis leads to extensive mucosal necrosis, denuded villi, and hemorrhage.

Downloaded from www.pdf.tube

its target antigen in fixed tissue sections. This antigen-antibody complex is then visualized using enzymatic reactions that precipitate a colored chromogen at the site of binding.

208. (A) P-3, Q-2, R-1, S-4
The Ziehl-Neelsen stain is used to identify acid-fast bacteria like Mycobacterium. Grocott's methenamine silver stain reacts with carbohydrates to highlight fungal walls. Mucicarmine is highly specific for staining the polysaccharide capsule of Cryptococcus species. The Warthin-Starry silver stain is a classic technique used to visualize spirochetes, such as Leptospira, in tissue sections.

NODIA

209. (B) Antigen retrieval → Primary antibody incubation → Secondary antibody incubation → Chromogen application
In standard indirect immunohistochemistry, formalin fixation creates protein cross-links that can mask tissue antigens. Therefore, the first essential step is antigen retrieval to restore immunoreactivity. This is followed by incubation with a primary antibody, then a secondary antibody that binds the primary antibody. Finally, a chromogen is applied to visualize the localized reaction.

210. (C) Mycolic acid in their cell wall
The biological basis of acid-fastness in Mycobacteria is the unique, complex composition of their cell wall. It contains high concentrations of long-chain fatty acids, specifically mycolic acids. These hydrophobic lipids form a dense barrier that binds the primary stain, carbol fuchsin. This strong bond resists decolorization by acid-alcohol, allowing the bacilli to retain the red color.

****

lymphatics, causing severe interlobular septal edema. Concurrently, adjacent lung lobules show different stages of hepatization, consolidation, and congestion, producing a variegated, marbled macroscopic pattern in lungs.

161. (A) Lacks the pronounced interlobular septal marbling
Contagious Caprine Pleuropneumonia is caused by Mycoplasma capricolum subsp. capripneumoniae. While the disease resembles bovine pleuropneumonia due to severe fibrinous pleuritis and hepatization, the caprine lung lacks prominent interlobular septa. Consequently, caprine lesions are more homogenous and do not exhibit the pronounced interlobular septal marbling characteristic of bovine pulmonary infections.

162. (C) Mycoplasma gallisepticum
Chronic respiratory disease in poultry is primarily initiated by Mycoplasma gallisepticum. The bacterium targets the mucosal surfaces of the upper respiratory tract, trachea, and air sacs. The infection is characterized by progressive mucosal thickening, loss of ciliary function, and mild lymphofollicular infiltration, which often becomes complicated by secondary bacterial pathogens.

163. (A) Thickened air sacs with caseous exudate
Uncomplicated avian mycoplasmosis is typically mild, but field cases are frequently complicated by Escherichia coli. This mixed infection causes severe systemic lesions. The most characteristic pathological findings in birds with complicated disease include severe thickening and cloudiness of the air sacs, which become filled with yellow, caseous inflammatory exudate in the body.

164. (D) Pleura, pericardium, and peritoneal cavity
Specific invasive strains of Mycoplasma can enter the host vascular system, leading to systemic dissemination. This bacteremia often leads to the development of fibrinous polyserositis in young production animals. The disease is characterized by thick, yellow sheets of fibrin deposited over the surfaces of the pleura, pericardium, and peritoneal membranes in these cavities.

165. (A) Both A and R are true and R is the correct explanation of A.
Mycoplasmas are distinct from typical bacteria due to evolutionary gene reduction. They do not possess a rigid peptidoglycan cell wall, instead maintaining cellular integrity with a triple-layered cell membrane. Because beta-lactam antibiotics act exclusively by inhibiting bacterial cell wall peptidoglycan synthesis, these organisms are completely refractory and resistant to penicillin therapy in animals.

166. (C) Necrotic lung tissue walled off by a fibrous capsule
In chronic stages of Contagious Bovine Pleuropneumonia, the host immune response attempts to isolate areas of extensive pulmonary infarction. This focal necrotic lung tissue is surrounded and walled off by a dense, thick capsule of fibrous connective tissue. This structural lesion, containing a dead piece of consolidated lung, is referred to as a sequestrum.

167. (D) Synovitis with purulent joint and sheath exudate
Mycoplasma synoviae exhibits a strong tissue tropism for the synovial membranes of joints and tendon sheaths in poultry. The systemic infection triggers an acute to chronic localized inflammatory response. Pathologically, this manifests as severe, exudative synovitis, where the joint spaces and surrounding tendon sheaths become distended with a thick, yellow-gray purulent exudate.

168. (A) Their small size and lack of cell wall make them invisible
Mycoplasmas are the smallest self-replicating prokaryotes, with dimensions ranging from one hundred to three hundred nanometers. Because they lack a cell wall and fail to stain with aniline dyes, they cannot be resolved as individual bacteria under a light microscope using standard hematoxylin and eosin staining. Identification requires specialized stains, immunohistochemistry, or molecular methods.

169. (B) Vascular endothelial cells
Ehrlichia ruminantium is an obligate intracellular bacterium that is transmitted to ruminant hosts by Amblyomma ticks. Once in the host, the organism shows a distinct, highly specific tropism for vascular endothelial cells, particularly within the brain, lungs, and kidneys. Replication within the cytoplasm of these host cells forms microcolonies, causing endothelial dysfunction.

Click Here to Buy book on Flipkart

170. (B) Pronounced accumulation of fluid in the pericardium
The vascular endothelial injury caused by replication of Ehrlichia ruminantium leads to a significant increase in vascular permeability throughout the host body. This vascular leakage results in widespread transudation of protein-rich fluid into body cavities. The most striking and classic gross pathological finding is the accumulation of clear, straw-colored fluid within the pericardial sac.

171. (A) Replication within red blood cells causing extravascular hemolysis
Anaplasma marginale is an obligate intracellular rickettsial pathogen of ruminants. It infects host erythrocytes, replicating as small membrane-bound inclusions at the cell periphery. The presence of the parasite alters the host red blood cell membrane, leading to their recognition and rapid phagocytosis by splenic and hepatic macrophages, resulting in severe extravascular hemolytic anemia.

172. (C) Widespread vasculitis, hemorrhage, and pancytopenia
Ehrlichia canis is the etiologic agent of canine monocytic ehrlichiosis. In the acute stage, the organism targets circulating monocytes and tissue macrophages. The infected cells migrate throughout the host, inducing generalized immunological vasculitis. This systemic vascular injury leads to petechial hemorrhages, platelet consumption, bone marrow suppression, and progressive pancytopenia in infected dogs.

173. (D) Morulae clusters in the cytoplasm of endothelial cells
In animals with suspected Heartwater, cytological diagnosis relies on identifying Ehrlichia ruminantium within its specific host target cells. Microscopic examination of brain smear preparations, particularly from the cerebral or cerebellar cortex, highlights host vascular capillaries. Within the cytoplasm of these capillary endothelial cells, the bacteria appear as distinct clusters of coccoid structures called morulae.

174. (C) Absence of hemoglobinuria despite severe anemia
Anaplasmosis and Babesiosis both cause severe anemia in cattle, but their pathological mechanisms differ. Babesiosis causes rapid intravascular hemolysis, releasing free hemoglobin that is excreted in the urine. Conversely, Anaplasma marginale causes purely extravascular hemolysis, as infected red cells are slowly removed by phagocytes. This prevents free hemoglobin release, ensuring the complete absence of hemoglobinuria.

175. (A) P-1, Q-2, R-3, S-4
Anaplasma marginale is an obligate intracellular rickettsial organism that targets bovine erythrocytes. Ehrlichia canis exhibits a tropism for host monocytes and macrophages, causing canine monocytic ehrlichiosis. Ehrlichia ruminantium infects endothelial cells, leading to increased vascular permeability in ruminants. Anaplasma platys is a unique rickettsia that replicates within host canine platelets.

176. (D) Neutrophils and eosinophils
Anaplasma phagocytophilum is the tick-borne rickettsial pathogen responsible for causing tick-borne fever in ruminants. Unlike other rickettsial species that target red blood cells or endothelial cells, this pathogen has a distinct affinity for phagocytic cells. It selectively infects and replicates within mature and incomplete granulocytes, primarily targeting host neutrophils and eosinophils.

177. (A) Endothelial damage causing increased vascular permeability
In cases of Heartwater, the extensive replication of Ehrlichia ruminantium inside capillary endothelial cells causes physical disruption and functional impairment of the blood vessels. This endothelial damage destroys the vascular barrier, leading to a massive increase in capillary permeability. Protein-rich plasma leaks into interstitial spaces, causing generalized edema, hydrothorax, and ascites.

178. (C) Phagocytosis of infected, opsonized red cells by macrophages
The anemia associated with bovine anaplasmosis is primarily mediated by the immune system. When red blood cells are infected with Anaplasma marginale, the host produces antibodies that bind to the parasite antigens on the cell membrane. These opsonized erythrocytes are recognized as abnormal and are phagocytosed and destroyed by mononuclear macrophages in the spleen.

NODIA

179. (A) Elementary body
Chlamydia species possess a unique developmental cycle characterized by two distinct structural forms. The elementary body represents the extracellular, infectious stage of the parasite. This metabolic-dormant form is small and possesses a rigid, highly cross-linked outer membrane that allows it to survive in the extracellular environment and attach to susceptible host epithelial cells.

180. (D) Reticulate body
Following host cell entry, the infectious Chlamydial elementary body reorganizes into the reticulate body. This structural form is larger, fragile, and lacks the cross-linked membrane of the infectious form. The reticulate body is metabolically active and replicates rapidly via binary fission inside the membrane-bound cytoplasmic vacuole, utilizing the host cell energy resources.

181. (B) Necrotizing placentitis of cotyledons and intercotyledonary areas
Enzootic abortion of ewes is initiated when Chlamydia abortus targets the gestational uterus. The bacterium localizes and replicates inside trophoblasts, causing extensive cell lysis. The resulting gross lesions are characterized by severe, necrotizing placentitis that involves both the cotyledons and the adjacent intercotyledonary tissues, which appear thickened, clay-colored, and covered in yellow-gray exudate.

182. (A) Both A and R are true and R is the correct explanation of A.
Avian chlamydiosis is a systemic disease caused by Chlamydia psittaci. The bacterium infects host macrophages, disseminating throughout the vascular system. This systemic replication damages endothelial cells and activates inflammatory cascades. This vascular damage leads to severe, widespread exudation of protein-rich, fibrin-containing fluid, manifesting pathologically as pronounced fibrinous polyserositis involving the pleura, pericardium, and liver.

183. (D) Marked splenomegaly, hepatomegaly, and fibrinous pericarditis
Psittacosis in avian hosts is characterized by systemic infection of macrophages and subsequent hematogenous spread. The resulting pathology is dominated by severe systemic inflammation. Gross examination of affected birds classically reveals marked enlargement of the spleen and liver, accompanied by a thick layer of yellow fibrinous exudate covering the heart and surrounding pericardial sac.

184. (A) Modified Ziehl-Neelsen or Gimenez stain method
In histopathological sections, Chlamydiae are difficult to visualize using routine hematoxylin and eosin staining due to their minute size. However, their unique cell wall structure allows them to retain specific dyes. The modified Ziehl-Neelsen or Gimenez staining techniques highlight these intracellular microcolonies, rendering the inclusions bright red against a contrasting blue or green background.

185. (A) Attachment of EB → Internalization → Conversion to RB → Multiplication of RB → Conversion to EB → Cell lysis and release
The chlamydial life cycle begins when the infectious elementary body attaches to host cells, triggering endocytosis. Once inside, the elementary body reorganizes into the larger, metabolically active reticulate body. This form replicates via binary fission within inclusions. Eventually, the reticulate bodies condense back into elementary bodies, which are released upon host cell lysis.

NODIA

186. (B) Polyarthritis and conjunctivitis
Apart from causing reproductive failures, Chlamydia species can cause systemic diseases in sheep, goats, and cattle. In young growing animals, systemic dissemination can lead to deposition of organisms in synovial membranes and ocular tissues. This results in the development of severe, painful polyarthritis, accompanied by persistent bilateral keratoconjunctivitis and ocular discharge.

187. (A) Brucella abortus
The placental pathology in sheep infected with Chlamydia abortus is characterized by severe necrotizing placentitis. These gross and microscopic changes are virtually indistinguishable from the lesions produced by Brucella abortus in cattle. Both pathogens cause extensive necrosis of the cotyledons, accompanied by marked thickening and leathery yellow discoloration of the intercotyledonary chorion.

132. (D) Striated muscles including the heart, masseter, and tongue
Ingestion of Taenia saginata eggs by cattle leads to the release of oncospheres in the intestine. These larvae enter the bloodstream and distribute throughout the body, showing a strong predilection for active striated muscles. They encyst as fluid-filled larvae called Cysticercus bovis, which are commonly found in the masseter, tongue, and cardiac muscles.

133. (B) Taenia solium
Neurocysticercosis is a severe disease occurring when humans ingest eggs of Taenia solium, usually via fecal-oral contamination. The oncospheres migrate to the central nervous system and develop into fluid-filled larval cysts called Cysticercus cellulosae. Their presence in the brain parenchyma or ventricular system can cause chronic inflammation, seizures, and other neurological deficits.

134. (C) Mechanical irritation by flukes and toxic excretion products
The development of severe biliary epithelial hyperplasia in chronic liver fluke infections is a multi-factorial process. The physical movement of adult flukes, combined with the abrasive action of their spiny teguments, causes mechanical injury. Additionally, the release of metabolic excretory-secretory products, particularly proline, directly stimulates the proliferation of biliary epithelial cells.

135. (A) P-3, Q-1, R-2, S-4
Adult Fasciola hepatica reside in the bile ducts, causing periductal fibrosis known as pipe-stem liver. Echinococcus granulosus forms hydatid cysts in intermediate hosts. Larvae of Taenia solium encyst in swine muscles, producing the condition called measly pork. Moniezia expansa is a large tapeworm of ruminants that causes minimal clinical pathology in the gut.

136. (A) Protoscoleces
The inner lining of a fertile hydatid cyst consists of an active germinal layer that undergoes asexual reproduction. This layer buds off brood capsules, which subsequently produce multiple microscopic structures called protoscoleces. These represent the infective stage of the parasite and accumulate within the cyst fluid, along with free-floating daughter cysts and capsule debris.

137. (B) A small, oval vesicle containing a single invaginated scolex
Cysticercosis is characterized by the presence of larval tapeworm stages in tissues. Macroscopically, these individual lesions appear as small, fluid-filled oval vesicles embedded within host muscles. Microscopic examination of a viable cyst reveals that it contains a single, invaginated scolex equipped with suckers and hooks, which is capable of everting upon ingestion by the definitive host.

138. (B) Duodenal migration of immature flukes causing hemorrhagic enteritis
While adult rumen flukes live attached to the ruminal wall causing minimal damage, the immature stages are highly pathogenic. When young livestock ingest metacercariae, these larvae excyst and penetrate the duodenal mucosa. Their active migration through the proximal small intestinal wall causes severe hemorrhagic enteritis, mucosal erosion, hypoproteinemia, and profuse diarrhea.

139. (A) Babesia bovis
Babesia species replicate inside host red blood cells, eventually causing cell lysis. In acute infections, this rapid replication leads to massive, widespread intravascular hemolysis. The resulting clinical and pathological hallmarks include severe regenerative anemia, icterus, and hemoglobinuria, which is characterized by the accumulation of dark red urine containing free hemoglobin.

140. (C) Dark red to black kidneys from hemoglobin cast formation
In acute cases of bovine babesiosis, the massive lysis of infected erythrocytes releases large quantities of free hemoglobin into the blood. This free hemoglobin is filtered by the renal glomeruli, overwhelming the resorptive capacity of the tubules. Consequently, hemoglobin accumulates in the tubular lumen, forming prominent casts that turn the kidneys dark red or black.

141. (D) Aggregation of infected erythrocytes in brain capillaries
Babesia bovis infections often manifest with severe neurological signs. This syndrome is driven by cytoadherence, where infected erythrocytes express parasite-derived proteins on their surfaces that bind to vascular endothelial cells. This adhesion leads to sequestration and clustering

Downloaded from www.pdf.tube

198. (A) P-2, Q-1, R-3, S-4
Scrapie is a transmissible spongiform encephalopathy affecting sheep and goats. Bovine Spongiform Encephalopathy is the classic prion disease of cattle. Chronic Wasting Disease is unique because it infects wild and captive cervids, including deer and elk. Transmissible Mink Encephalopathy is a rare prion disease occurring in farmed mink populations worldwide.

199. (D) An inflammatory immune cell response
Transmissible spongiform encephalopathies cause severe progressive neurological damage, but their pathology is distinct from viral encephalitides. While viral infections trigger prominent perivascular cuffing by lymphocytes and microgliosis, prion diseases conspicuously lack any inflammatory response. The host immune system does not recognize the misfolded prions as foreign, preventing lymphocyte or neutrophil infiltration.

200. (A) Medulla oblongata at the level of the obex
In veterinary diagnostic pathology, confirming scrapie in sheep relies on histopathological examination of the brain. The neuroanatomical lesions are not uniformly distributed. The medulla oblongata, specifically at the level of the obex, is the most consistent site for finding early and prominent neuronal vacuolation, making it the mandatory regulatory diagnostic site.

201. (B) II
Scrapie is characterized clinically by intense pruritus, leading to wool loss from rubbing. Pathologically, transmissible spongiform encephalopathies are completely non-inflammatory, showing no immune cell infiltration. Prominent features include astrogliosis, where astrocytes proliferate and hypertrophy in response to neuronal death. These diseases are invariably fatal, with no recorded survival once symptoms appear.

202. (B) Neuronal cytoplasm (perikaryon) and neuropil
The characteristic vacuoles observed in brain sections of animals with transmissible spongiform encephalopathies are localized to specific structures. Under high-power magnification, these clear vacuoles are situated primarily within the neuronal cytoplasm, specifically the perikaryon. Vacuolation also extensively affects the neuronal processes within the neuropil, producing the classic spongiform tissue pattern.

203. (A) Pathogenic prion protein ($\mathrm{Pr}\,P^{\{Sc\}}$)
While histopathology is highly suggestive, definitive diagnosis of transmissible spongiform encephalopathies requires identifying the causative agent. Immunohistochemistry is the gold standard for this purpose. This technique utilizes specific monoclonal antibodies to bind and highlight the accumulation of the misfolded, protease-resistant pathogenic prion protein within the brain tissues of affected host animals.

204. (C) Acid-fast Mycobacterium bacilli
In diagnostic veterinary pathology, the Ziehl-Neelsen stain is a specialized histochemical technique. It is utilized to demonstrate acid-fast organisms, most notably Mycobacterium species. The high lipid content in their cell walls resists decoloring by acid-alcohol. Consequently, these bacilli retain the primary red dye, appearing as bright red rods against a blue background.

NODIA

205. (A) Both A and R are true and R is the correct explanation of A.
The Periodic Acid-Schiff stain is a valuable diagnostic tool in pathology. Periodic acid oxidizes adjacent glycol groups in carbohydrates to form aldehydes. These aldehydes react with Schiff's reagent, producing a brilliant magenta color. Because fungal cell walls are composed of complex polysaccharides, this stain highlights fungal structures, making them easily identifiable in tissue.

206. (C) Dark brown to black
Grocott's methenamine silver stain is a highly sensitive histochemical method for visualizing fungal elements in tissue sections. The procedure utilizes chromic acid to oxidize the carbohydrates in fungal cell walls, generating aldehydes. These aldehydes then reduce the methenamine silver complex, depositing metallic silver which renders the fungi dark brown or black against a green background.

207. (B) An antibody and its specific host tissue antigen
Immunohistochemistry is a powerful diagnostic technique that combines anatomical, immunological, and biochemical principles. The fundamental mechanism of this procedure is the highly specific binding between an antibody and

151. (D) Focal necrosis and calcification of cotyledons
Toxoplasma gondii infection in pregnant ewes often leads to gestational failure. The parasite targets the placental tissues, where it replicates and causes focal necrosis within the cotyledons. Macroscopic examination of the placenta reveals characteristic small, white, gritty spots on the cotyledons, which represent areas of focal necrosis accompanied by secondary dystrophic calcification.

152. (A) Non-suppurative encephalomyelitis with focal necrosis
Neosporosis is a major cause of abortion in cattle. In infected fetuses, the parasite exhibits a strong tissue tropism for the central nervous system. Histopathological examination of fetal brain tissue reveals non-suppurative encephalomyelitis, which is characterized by prominent perivascular cuffing by mononuclear cells, focal glial nests, and small zones of focal parenchymal necrosis.

153. (B) Cryptosporidium parvum
Cryptosporidium parvum is a tiny protozoan parasite that infects the intestinal tract of neonatal calves. The organisms reside in a unique intracellular but extracytoplasmic position, located within the host microvillous brush border. This localization causes physical loss of microvilli and mild enterocyte damage, leading to malabsorptive diarrhea without deep mucosal invasion.

154. (B) Striated skeletal and cardiac musculature
Sarcocystis species require a two-host life cycle, where carnivores act as definitive hosts and herbivores serve as intermediate hosts. In the intermediate host, the asexual stages migrate to muscles. They develop into highly organized, slow-growing cysts containing numerous bradyzoites within striated skeletal and cardiac muscle fibers, which are normally clinically asymptomatic.

155. (A) I, II, III and IV
Felines serve as the sole definitive host for Toxoplasma gondii. Neospora caninum is a primary cause of bovine abortion worldwide. Eimeria stiedae causes distinct biliary hyperplasia and hepatic coccidiosis in rabbits. Sarcocystis cysts can be microscopic or macroscopic. However, Cryptosporidium species reside strictly within the microvillous border, never invading deep lamina propria.

156. (C) Enlarged bile ducts with papillary hyperplasia and oocysts
Eimeria stiedae specifically infects the epithelial cells of the biliary system in rabbits. The replication of the parasite stimulates marked mitotic activity in host cells. This results in severe, diffuse papillary hyperplasia of the bile duct epithelium. Affected bile ducts become visibly dilated and thickened, containing numerous developing oocysts and inflammatory debris.

157. (D) Central nervous system and skeletal muscle
Following the acute tachyzoite phase of Toxoplasma gondii, the host immune response limits rapid replication. The parasite then transforms into slowly replicating bradyzoites. These bradyzoites form highly stable, persistent tissue cysts within specific host organs. These dormant cysts are most commonly found in the brain, spinal cord, cardiac muscle, and skeletal muscle.

158. (C) Mild to moderate villous atrophy and fusion
Cryptosporidiosis in neonatal calves damages the small intestinal mucosa. The attachment of the organisms to the enterocytes causes loss of microvilli and disrupts normal cellular structure. Consequently, the affected intestinal villi undergo fusion and mild to moderate atrophy. This architectural change significantly decreases the absorptive surface area, causing watery, malabsorptive diarrhea.

159. (B) Fibrinous bronchopneumonia with marbling and pleuritis
Contagious Bovine Pleuropneumonia is a severe respiratory disease of cattle. The pulmonary lesions are unilateral and localized to the diaphragmatic lobes. Grossly, the affected lungs exhibit severe fibrinous bronchopneumonia accompanied by marked thickening of the interlobular septa and fibrinous pleuritis. This classic pattern of septal edema and parenchymal consolidation creates a striking marbled appearance.

160. (D) Interlobular septal edema and variable lobular consolidation
The pathognomonic marbling of pulmonary tissues in bovine mycoplasmal pleuropneumonia is caused by distinct anatomical changes. The bacterial infection triggers profound inflammatory exudation into the interlobular

Click Here to Buy book on Amazon

CHAPTER 11
Avian Pathology

2015

1. Which of the following is the standard anatomical location to initiate the primary incision during a systematic avian necropsy?
(A) Dorsal midline from the occiput to the uropygial gland
(B) Mid-ventral line from the intermandibular space to the cloaca
(C) Ventral abdominal wall immediately posterior to the sternum
(D) Bilateral incisions along the medial aspect of the thigh

2021

2. What is the most appropriate fixative ratio for routine histological processing of avian tissues to prevent autolysis?
(A) 1:2 tissue to fixative ratio
(B) 1:5 tissue to fixative ratio
(C) 1:10 tissue to fixative ratio
(D) 1:20 tissue to fixative ratio

2002

3. Match the avian lymphoid organs in List I with their primary functional characteristics or locations in List II.
List I: Lymphoid Organs
P. Bursa of Fabricius
Q. Thymus
R. Spleen
S. Harderian gland
List II: Characteristics or Locations
1. Lobulated organ parallel to the vagus nerve
2. Primary site for B-cell maturation
3. Paraocular aggregation of plasma cells
4. Major secondary lymphoid organ lacking cortex and medulla
(A) P-2, Q-1, R-4, S-3
(B) P-1, Q-2, R-4, S-3
(C) P-2, Q-4, R-1, S-3
(D) P-4, Q-1, R-2, S-3

2025

4. During post-mortem examination, a thickened, edematous, and hyperemic Bursa of Fabricius covered with a gelatinous transudate is most indicative of which stage of Infectious Bursal Disease?
(A) Chronic phase (day 10 post-infection)
(B) Acute phase (day 3-4 post-infection)
(C) Recovery phase (day 14 post-infection)
(D) Subclinical phase

2010

5. The Harderian gland in poultry is primarily responsible for the local synthesis and secretion of which immunoglobulin class in the ocular/respiratory mucosa?
(A) IgA
(B) IgY
(C) IgM
(D) IgD

2006

6. Identify the correct combination of statements regarding early chick mortalities.
I. Omphalitis is typically a non-contagious condition primarily caused by opportunistic bacterial infections.
II. Yolk sac infection strictly occurs due to vertical transmission of pathogens from the hen.
III. 'Navel ill' frequently results from improper incubator hygiene and high humidity.
Which of the above statements are correct?
(A) I and II
(B) I and III
(C) II and III
(D) I, II and III

2002

7. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option.
Assertion (A): Retained yolk sacs are frequently observed in chicks suffering from omphalitis.
Reason (R): Bacterial proliferation in the umbilicus produces toxins that accelerate the physiological absorption of the yolk sac.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.

NODIA

2005

26. Which of the following is the causative agent of Fowl Typhoid, producing bronze discoloration of the liver in mature poultry?
(A) Salmonella Gallinarum
(B) Salmonella Enteritidis
(C) Salmonella Pullorum
(D) Salmonella Typhimurium

2023

27. Determine the correct chronological arrangement of the pathogenic events in systemic Colibacillosis (APEC infection) starting from inhalation.
I. Colonization of the air sacs leading to airsacculitis
II. Fibrinous pericarditis and perihepatitis
III. Inhalation of E. coli-contaminated dust
IV. Septicemia and systemic dissemination
Select the correct sequence from the options below.
(A) III, I, IV, II
(B) III, IV, I, II
(C) I, III, II, IV
(D) IV, III, I, II

2003

28. In Avian Mycoplasmosis (CRD), the exudate found in the air sacs typically progresses from serous to which morphological state in chronic, complicated cases?
(A) Purulent
(B) Caseous/Cheesy
(C) Hemorrhagic
(D) Gelatinous

2025

29. Fowl cholera, caused by Pasteurella multocida, often manifests in its chronic form with localized infections. Which of the following is a classic site for this localized chronic lesion?
(A) The wattles
(B) The central nervous system
(C) The uropygial gland
(D) The proventriculus

2012

30. Which staining technique is most appropriate to demonstrate the bipolar staining characteristics of Pasteurella multocida from a blood smear in acute Fowl Cholera?
(A) Gram staining technique
(B) Ziehl-Neelsen staining technique
(C) Leishman's or Methylene blue stain
(D) Periodic Acid-Schiff staining technique

2001

31. Avian chlamydiosis (Ornithosis) is a zoonotic disease caused by Chlamydia psittaci. Which gross lesion is classically observed during necropsy of infected parrots or turkeys?
(A) Severe hemorrhagic tracheitis with mucosal ulceration
(B) Vesicular lesions on the comb, wattles, and eyelids
(C) Necrotic enteritis in the jejunum and ileal region
(D) Fibrinous serositis (pericarditis and airsacculitis)

2024

32. Avian spirochaetosis is primarily transmitted by which of the following biological vectors?
(A) Culicoides midges
(B) Dermanyssus gallinae (red mite)
(C) Argas persicus (fowl tick)
(D) Simulium flies

2006

33. The classical gross lesions of "brooder pneumonia" (Aspergillosis) in young chicks primarily involve the presence of:
(A) Fibrinous casts in the tracheal lumen and bronchi
(B) Yellow-white caseous nodules in the lungs and air sacs
(C) Petechial hemorrhages on the epicardium and myocardium
(D) Necrotic foci in the liver parenchyma and spleen

2014

34. Crop mycosis, characterized by a "terry-towel" appearance of the crop mucosa, is primarily caused by which opportunistic fungal pathogen?
(A) Aspergillus fumigatus
(B) Histoplasma capsulatum
(C) Cryptococcus neoformans
(D) Candida albicans

2012

35. In chronic aflatoxicosis in poultry, the liver often exhibits which of the following pathological changes?
(A) Fatty change, biliary hyperplasia, and fibrosis
(B) Acute massive necrosis and hemorrhage
(C) Granulomatous inflammation with giant cells
(D) Suppurative hepatitis

Downloaded from www.pdf.tube

is the frequent involvement of which of the following tissues in Marek's disease?
(A) Liver
(B) Peripheral nerves
(C) Spleen
(D) Bursa of Fabricius

2003
18. Histologically, Avian Leukosis Virus (ALV) tumors are characterized by homogeneous populations of which type of cells?
(A) Pleomorphic T-lymphocytes
(B) Macrophages and multinucleated giant cells
(C) Mature plasma cells and active heterophils
(D) Lymphoblasts and uniformly large B-cells

2012
19. Identify the correct combination of statements concerning avian oncogenic viruses.
I. Marek's disease is caused by a Gallid alphaherpesvirus 2.
II. Avian leukosis virus typically induces nodular tumors exclusively in birds older than 14 weeks.
III. Marek's disease tumors are strictly composed of B-lymphocytes.
IV. Ocular lymphomatosis is a classical manifestation of Marek's disease.
V. Both diseases vertically transmit at high rates through the egg.
Which of the above statements are correct?
(A) I, II and IV
(B) II, III and V
(C) I, III and IV
(D) I, IV and V

2009
20. Which adenoviral condition is characterized by a pathognomonic accumulation of straw-colored fluid in the pericardial sac and primarily affects broilers aged 3 to 5 weeks?
(A) Inclusion body hepatitis
(B) Egg drop syndrome
(C) Hydropericardium syndrome
(D) Marble spleen disease

2005
21. Egg Drop Syndrome-76 (EDS-76) is caused by an aviadenovirus primarily transmitted through which of the following natural hosts?
(A) Waterfowl
(B) Turkeys
(C) Pigeons
(D) Quails

2009
22. In Inclusion Body Hepatitis (IBH), the hallmark microscopic lesion in the hepatocytes is the presence of:
(A) Intracytoplasmic, eosinophilic Negri bodies
(B) Large, acidophilic intracytoplasmic Guarnieri bodies
(C) Small, basophilic nuclear remnants of Howell-Jolly bodies
(D) Basophilic or eosinophilic intranuclear inclusion bodies

2009
23. Arrange the chronological steps of pathogenesis in Avian Reovirus-induced tenosynovitis.
I. Viral replication in the enteric tract and bursa
II. Inflammation of the gastrocnemius tendon sheath
III. Oral-fecal exposure to the reovirus
IV. Fibrosis and eventual rupture of the tendon
V. Transient viremia
Select the correct sequence from the options below.
(A) III, I, V, II, IV
(B) I, III, II, V, IV
(C) III, V, I, IV, II
(D) V, III, I, II, IV

NODIA

2006
24. Malabsorption syndrome in poultry, often associated with reovirus infections, presents with a characteristic gross lesion in the proventriculus described as:
(A) Hemorrhagic banding across the proventricular tonsils
(B) Necrotic ulceration of the mucosal and glandular layers
(C) Proventricular enlargement with pale, flaccid walls
(D) Fibrinous exudate filling the entire glandular lumen

2011
25. Which characteristic pathological finding is most strongly associated with Pullorum disease in adult carrier hens?
(A) Massive splenomegaly with a prominent mottled appearance
(B) Hemorrhagic enteritis involving the lower duodenal loop
(C) Thickened, granular crop mucosa with diphtheritic plaques
(D) Misshapen, discolored, and pedunculated ovarian follicles

(B) Riboflavin (B2)
(C) Niacin (B3)
(D) Pyridoxine (B6)

2015

47. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option.

Assertion (A): Vitamin A deficiency in poultry leads to the formation of pustule-like lesions in the esophagus and crop.

Reason (R): Squamous metaplasia of the mucous gland epithelium occurs due to hypovitaminosis A, blocking the ducts and forming distended retention cysts.

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2021

48. Exudative diathesis, characterized by severe subcutaneous edema of the breast and abdomen, results from a simultaneous deficiency of Vitamin E and which trace mineral?
(A) Zinc
(B) Copper
(C) Selenium
(D) Manganese

2017

49. In formulating poultry diets, an inverted Calcium to Phosphorus ratio (e.g., higher P than Ca) heavily predisposes growing birds to which skeletal pathology?
(A) Generalized osteopetrosis with cortical thickening
(B) Nutritional secondary hyperparathyroidism (Rickets)
(C) Tibial dyschondroplasia with retained cartilage plugs
(D) Spondylolisthesis with spinal cord compression

2023

50. Perosis (slipped tendon) in growing birds is anatomically characterized by the displacement of the gastrocnemius tendon from the intercondylar groove. It is fundamentally linked to a deficiency of:
(A) Iron and copper
(B) Calcium and Vitamin D3
(C) Magnesium and potassium
(D) Manganese and choline

2005

51. Identify the correct combination of statements concerning avian skeletal pathologies.
I. Rickets in young birds features poorly mineralized, widened growth plates.
II. Cage layer fatigue is characterized by excessive medullary bone formation.
III. Tibial dyschondroplasia involves avascular plugs of cartilage in the proximal tibiotarsus.
Which of the above statements are correct?
(A) I and II
(B) I and III
(C) II and III
(D) I, II and III

2005

52. Ascites syndrome (Pulmonary Hypertension Syndrome) in fast-growing broilers culminates in right ventricular failure. Which gross lesion is a definitive indicator of this terminal right-sided heart failure?
(A) Fibrinous pericarditis with extensive epicardial adhesion
(B) Endocardiosis of the left AV valve with atrial dilation
(C) Marked right ventricular hypertrophy and dilation
(D) Severe pulmonary hemorrhages with generalized congestion

2015

53. Round heart disease is a spontaneously occurring cardiomyopathy predominantly observed in which avian species?
(A) Turkeys
(B) Chickens
(C) Ducks
(D) Geese

2002

54. In the pathogenesis of visceral gout, the chalky white deposits found on the pericardium and liver capsule consist primarily of:
(A) Calcium oxalate crystals
(B) Cholesterol clefts
(C) Calcium pyrophosphate
(D) Monosodium urate crystals

2017

55. Identify the incorrect combination of statements regarding avian renal pathology.
I. Articular gout generally occurs in birds with normal blood uric acid levels but localized trauma.

Click Here to Buy book on Flipkart

2025
36. Ochratoxin A, a mycotoxin produced by Aspergillus and Penicillium species, primarily targets which organ system in poultry, leading to swelling and urate deposition?
(A) Hepatic system
(B) Central nervous system
(C) Renal system
(D) Cardiovascular system

2002
37. Fusariotoxicoses, caused by toxins such as trichothecenes (e.g., T-2 toxin), classically produce which distinct lesion in poultry?
(A) Oral and proventricular mucosal necrosis (oral lesions)
(B) Caecal core formation with extensive mucosal sloughing
(C) Demyelination of peripheral nerves in the sciatic plexus
(D) Ovarian follicle regression and severe internal laying

2010
38. Which Eimeria species is known for causing severe hemorrhagic lesions specifically in the ceca of chickens?
(A) Eimeria acervulina
(B) Eimeria maxima
(C) Eimeria necatrix
(D) Eimeria tenella

2004
39. A post-mortem of a broiler reveals ballooned mid-intestines with characteristic "salt and pepper" serosal lesions and hemorrhagic enteritis. The most likely causative agent is:
(A) Eimeria praecox
(B) Eimeria necatrix
(C) Eimeria brunetti
(D) Eimeria maxima

2023
40. Histomoniasis (Blackhead disease) produces pathognomonic target-like necrotic lesions in which organ of turkeys?
(A) Spleen
(B) Lungs
(C) Liver
(D) Kidneys

2005
41. The transmission of Histomonas meleagridis in poultry flocks is uniquely facilitated by its encystment within the ova of which nematode?
(A) Ascaridia galli
(B) Capillaria obsignata
(C) Syngamus trachea
(D) Heterakis gallinarum

2008
42. A massive infestation of Ascaridia galli is most likely to cause which of the following mechanical pathologies in chickens?
(A) Intestinal impaction or rupture
(B) Tracheal obstruction and asphyxia
(C) Biliary duct fibrosis and necrosis
(D) Esophageal stricture and dilatation

2003
43. Match the avian parasites in List I with their primary predilection sites in the host in List II.
List I: Parasites
P. Capillaria annulata
Q. Heterakis gallinarum
R. Raillietina cesticillus
S. Syngamus trachea
List II: Predilection Sites
1. Ceca
2. Small intestine
3. Trachea
4. Crop and esophagus
(A) P-4, Q-1, R-2, S-3
(B) P-1, Q-4, R-2, S-3
(C) P-3, Q-1, R-4, S-2
(D) P-4, Q-2, R-1, S-3

2002
44. The intermediate hosts essential for the transmission of the poultry cestodes Raillietina spp. are primarily:
(A) Earthworms and slugs
(B) Ants and beetles
(C) Snails and slugs
(D) Mosquitoes and midges

2015
45. Encephalomalacia ("crazy chick disease") is a neuropathological manifestation caused by the deficiency of which vitamin?
(A) Vitamin E
(B) Vitamin A
(C) Vitamin D3
(D) Vitamin K

2018
46. A flock of 3-week-old chicks presents with inward curling of the toes and birds resting on their hocks. Histopathology reveals swelling and demyelination of the sciatic nerves. This condition is caused by a deficiency of:
(A) Thiamine (B1)

Downloaded from www.pdf.tube

(A) Liver
(B) Brain
(C) Heart
(D) Pancreas

2014
64. A pendulous crop is a chronically distended, flaccid crop that fails to empty. While multicausal, a strong genetic predisposition for this condition exists heavily in which of the following?
(A) Single Comb White Leghorns
(B) Heavy-meat Cornish crosses
(C) Broad Breasted Bronze Turkeys
(D) Standard Japanese Quails

2005
65. Swollen head syndrome, featuring facial edema and purulent exudate in the subcutaneous tissues of the head, is classically a complicated infection initiated by which primary pathogen?
(A) Fowlpox virus
(B) Avian Metapneumovirus
(C) Avian Influenza Virus
(D) Mycoplasma synoviae

2023
66. Breast blisters (sternal bursitis) are an important cause of carcass downgrading in heavy broilers and turkeys. The fluid accumulation occurs in response to continuous pressure and friction against which anatomical structure?
(A) Coracoid bone of the shoulder
(B) Clavicle (furcula) bone of the chest
(C) Ischium bone of the pelvis
(D) Keel bone (sternum) of the breast

2014
67. Bumble foot (pododermatitis) involves chronic, granulomatous, and purulent inflammation of the footpad. The most frequently isolated bacterial agent from these lesions is:
(A) Staphylococcus aureus
(B) Streptococcus pneumoniae
(C) Pseudomonas aeruginosa
(D) Campylobacter jejuni

2016
68. Cannibalism in poultry is a behavioral vice that can lead to severe injury and mortality. Which of the following nutritional deficiencies is classically cited as a metabolic trigger for pecking behavior?
(A) Highly soluble simple carbohydrate deficiency
(B) Fat-soluble Vitamin K or phylloquinone deficiency
(C) Sodium and amino acid (methionine) deficiency
(D) Excess dietary calcium leading to metabolic alkalosis

NODIA

2025
69. High Pathogenicity Avian Influenza (HPAI) viruses typically possess multiple basic amino acids at the cleavage site of which critical surface glycoprotein?
(A) Hemagglutinin (HA)
(B) Neuraminidase (NA)
(C) Matrix 2 (M2)
(D) Non-structural 1 (NS1)

2012
70. Pathognomonic gross lesions of Highly Pathogenic Avian Influenza (HPAI) in chickens frequently include cyanosis and severe edema of the comb and wattles, alongside hemorrhages in which specific internal site?
(A) Within the medullary cavity of long bones
(B) Along the trunk of the sciatic nerve
(C) Inside the lymphoid tissue of bursa of Fabricius
(D) Between the proventriculus and gizzard

2001
71. In comparing the necropsy findings of Newcastle disease (velogenic viscerotropic) and Highly Pathogenic Avian Influenza, which lesion is highly prevalent in HPAI but generally less prominent in ND?
(A) Splenic necrosis and prominent white mottling
(B) Subcutaneous edema of the head and neck
(C) Hemorrhagic changes in the cecal tonsils
(D) Proventricular mucosal hemorrhages at gland tips

2003
72. Which of the following best defines "pathotyping" in the context of avian viruses?
(A) Determining the precise nucleotide sequence of the viral genome using sequencing
(B) Assessing the virus's environmental survivability and persistence on fomites
(C) Classifying the virus based on its virulence and disease manifestation in the host
(D) Identifying the specific cellular receptors the viral glycoproteins use for entry

2003
73. A 4-week-old broiler flock experiences 5% sudden mortality. Necropsy reveals pale, swollen, and friable livers with subcapsular hemorrhages. Intranuclear inclusion bodies are observed on

II. Nephritis/nephrosis syndrome can be triggered by infectious bronchitis virus (nephropathogenic strains).
III. Water deprivation followed by sudden rehydration strictly prevents visceral gout.
IV. Ureters in gout-affected birds are typically distended with white urate concretions.

Which of the above statements are incorrect?
(A) I and II
(B) I and III
(C) II and IV
(D) III and IV

2015

56. Fatty Liver Haemorrhagic Syndrome (FLHS) is most commonly recognized as a cause of sudden death due to fatal intra-abdominal hemorrhage in:
(A) Young, rapidly growing meat broilers
(B) Free-range, actively foraging cockerels
(C) Caged, heavy-producing laying hens
(D) Turkey poults undergoing rapid skeletal growth

2017

57. Fatty Liver and Kidney Syndrome (FLKS) in young chicks, characterized by lethargy, hypoglycemia, and pale, lipid-laden livers and kidneys, is primarily responsive to supplementation with:
(A) Vitamin C
(B) Calcium
(C) Methionine
(D) Biotin

2024

58. Cage layer fatigue, causing osteomalacia, paralysis, and fragile bones in high-producing caged hens, is fundamentally caused by:
(A) Depletion of structural cortical bone to maintain calcium for eggshells
(B) Viral destruction of active osteoblasts in the medullary cavity
(C) Toxic accumulation of heavy metals disrupting normal bone deposition
(D) Excess dietary phosphorus inhibiting calcium absorption in the gut

2006

59. Identify the correct combination of statements concerning avian reproductive tract disorders.
I. A 'false layer' ovulates normally, but the yolk drops into the peritoneal cavity due to infundibular dysfunction.
II. Egg peritonitis frequently involves secondary bacterial infection with Escherichia coli.
III. Salpingitis refers to the inflammation of the ovary.
IV. Internal layers eventually reabsorb all yolks without pathological consequence.
V. Mycoplasma gallisepticum is a known trigger for salpingitis in commercial layers.

Which of the above statements are correct?
(A) I, II and V
(B) I, III and IV
(C) II, IV and V
(D) I, II and III

2008

60. The term 'false layer' clinically describes a bird that exhibits secondary sexual characteristics and visits the nest box, yet fails to produce an egg. What is the most common viral etiology leading to this permanent cystic destruction of the right oviduct or functional left oviduct?
(A) Late exposure to Egg Drop Syndrome virus
(B) Early exposure to Infectious Bronchitis Virus
(C) Infection with Marek's disease virus
(D) Vertical transmission of Avian Encephalomyelitis

2023

61. Panting (hyperventilation) due to acute heat stress in poultry physiologically alters blood gas dynamics resulting in:
(A) Respiratory alkalosis
(B) Metabolic acidosis
(C) Metabolic alkalosis
(D) Respiratory acidosis

2024

62. Which pathophysiological consequence of severe heat stress contributes directly to the thinning of eggshells in laying hens?
(A) Decreased blood pH leading to systemic calcium binding in bones
(B) Hypertrophy of the shell gland cells due to local inflammation
(C) Reduction in blood bicarbonate levels due to respiratory alkalosis
(D) Excess synthesis of calcitriol inhibiting active transport in gut

2014

63. Blue comb, also referred to as pullet disease or avian monocytosis, is a syndrome historically characterized by cyanosis of the comb and wattles, severe dehydration, and focal necrosis in which organ?

Click Here to Buy book on Amazon

(C) A is true but R is false.
(D) A is false but R is true.

2009

8. Which of the following bacterial species is most commonly isolated from cases of coliform septicemia leading to unabsorbed yolk sacs in neonates?
(A) Salmonella Pullorum
(B) Pasteurella multocida
(C) Escherichia coli
(D) Clostridium perfringens

2017

9. Pathognomonic pinpoint hemorrhages on the tips of the proventricular glands are most characteristically associated with which pathotype of Newcastle Disease Virus?
(A) Asymptomatic enteric pathotype
(B) Lentogenic respiratory pathotype
(C) Mesogenic neurotropic pathotype
(D) Velogenic viscerotropic pathotype

2018

10. Which viral glycoprotein is primarily responsible for the attachment of the Newcastle disease virus to the host cell receptors?
(A) Hemagglutinin-neuraminidase (HN) protein
(B) Fusion (F) membrane-penetrating protein
(C) Matrix (M) assembly-associated protein
(D) Nucleocapsid (NP) genome-binding protein

2019

11. Identify the incorrect combination of statements regarding Infectious Bronchitis (IB) and Infectious Laryngotracheitis (ILT).
I. ILT is caused by an alphaherpesvirus.
II. IB predominantly affects the lower respiratory tract without causing reproductive issues.
III. Hemorrhagic tracheitis with expectoration of blood is pathognomonic for severe ILT.
Which of the above statements are incorrect?
(A) I and II
(B) II only
(C) III only
(D) I and III

2007

12. In diagnosing infectious laryngotracheitis (ILT), the presence of which histological finding in the tracheal mucosa is considered pathognomonic?
(A) Intracytoplasmic inclusion bodies in epithelium
(B) Intranuclear inclusion bodies (Cowdry type A)
(C) Syncytia formation in the proventricular glands
(D) Reticuloendothelial hyperplasia of lymphoid organs

2022

13. Which respiratory disease of poultry is characterized by wrinkled, misshapen eggs with thin, watery albumen due to specific viral targeting of the magnum and uterus?
(A) Infectious bronchitis
(B) Newcastle disease
(C) Avian influenza
(D) Avian pneumovirus

2006

14. A standard bursal grading system evaluates the severity of lesions in Infectious Bursal Disease. A bursa that is profoundly atrophied, resembling a small seed, generally represents which lesion score?
(A) Score 1
(B) Score 2
(C) Score 3
(D) Score 4

2006

15. Identify the incorrect combination of statements concerning the pathogenesis of Infectious Bursal Disease (IBD).
I. IBDV primarily replicates in the macrophages of the liver before viremia.
II. The target cells for IBDV are actively dividing IgM-bearing B lymphocytes.
III. Very virulent IBDV (vvIBDV) strains cause high mortality but do not deplete B cells.
IV. IBD-induced immunosuppression predisposes chickens to inclusion body hepatitis.
Which of the above statements are incorrect?
(A) I and III
(B) II and IV
(C) I, III and IV
(D) II, III and IV

NODIA

2006

16. Which secondary infection is most frequently exacerbated in broiler flocks that have suffered early immunosuppression from subclinical Gumboro disease?
(A) Fowl pox
(B) Avian encephalomyelitis
(C) Gangrenous dermatitis
(D) Marek's disease

2001

17. The primary distinction between the gross pathology of Marek's disease and Avian leukosis

histopathology. The most likely etiology belongs to the family:
(A) Herpesviridae
(B) Reoviridae
(C) Coronaviridae
(D) Adenoviridae

2021
74. Which of the following terms correctly defines the pathological accumulation of transudate in the peritoneal cavity of a bird?
(A) Ascites
(B) Anasarca
(C) Hydropericardium
(D) Hydrocephalus

2009
75. A classic histopathological finding in cases of Fowl Pox (cutaneous form) is the presence of large intracytoplasmic inclusion bodies within the hyperplastic epithelium known as:
(A) Guarnieri bodies
(B) Bollinger bodies
(C) Cowdry Type B bodies
(D) Joest-Degen bodies

2012
76. Fowl pox differs fundamentally from infectious laryngotracheitis (ILT) because:
(A) Fowl pox is a double-stranded DNA adenovirus, whereas ILT is an enveloped herpesvirus.
(B) Fowl pox inclusion bodies are intracytoplasmic, whereas ILT inclusion bodies are intranuclear.
(C) Fowl pox causes extensive cutaneous or neurological signs, whereas ILT is strictly respiratory.
(D) Fowl pox exclusively affects mature turkeys, whereas ILT is highly specific to young chickens.

2016
77. Which of the following conditions is an example of an ascending infection of the avian female reproductive tract?
(A) Egg peritonitis caused by Escherichia coli entering through the cloaca
(B) Ovarian transmission of Salmonella Gallinarum from infected parent stock
(C) Viral replication of EDS-76 in the infundibular and shell gland mucosa
(D) Congenital aplasia of the right oviduct leading to fluid accumulation

2021
78. In avian oncology, what is the primary role of the v-src oncogene?
(A) It acts as a tumor suppressor gene in Marek's disease to regulate neoplastic cell division.
(B) It induces caspase-mediated apoptosis in bursa-derived IgM-bearing cellular populations.
(C) It is a viral oncogene carried by Rous sarcoma virus that causes rapid transformation.
(D) It codes for a specific reverse transcriptase inhibitor targeting exogenous retroviruses.

2021
79. Avian Metapneumovirus (aMPV) is established as the primary etiological agent for which of the following diseases in turkeys?
(A) Bluecomb disease (coronaviral enteritis of turkeys)
(B) Coronaviral enteritis causing high mortality in poults
(C) Hemorrhagic enteritis caused by turkey adenovirus-3
(D) Turkey coryza (Turkey Rhinotracheitis)

2002
80. The diagnostic "dropping wing" paralysis or asymmetrical leg paralysis (classic "dancer's pose") in chickens is clinically pathognomonic for:
(A) Riboflavin deficiency
(B) Calcium tetany
(C) Marek's disease
(D) Avian Encephalomyelitis

2010
81. In Avian Encephalomyelitis (epidemic tremor), histopathology of the central nervous system classically demonstrates:
(A) Purulent meningoencephalitis with extensive perivascular cuffing
(B) Central chromatolysis of neurons and non-purulent encephalomyelitis
(C) Demyelination of the peripheral nerves with lymphoid infiltration
(D) Spongiform encephalopathy with vacuolation of the neuropil layers

2007
82. Which of the following is the hallmark lesion of Infectious Coryza caused by Avibacterium paragallinarum?
(A) Caseous nodules in the lower respiratory tract leading to consolidation of lungs
(B) Diphtheritic membranes in the upper tracheal lumen causing complete asphyxiation

Downloaded from www.pdf.tube

(C) Hemorrhagic enteritis in the duodenum and upper jejunum with mucosal sloughing
(D) Severe acute catarrhal inflammation of the upper respiratory tract with facial edema

2003

83. Match the infectious agents in List I with their corresponding typical target organs or tissues in List II.

List I: Infectious Agents
P. Eimeria acervulina
Q. Mycoplasma synoviae
R. Infectious Laryngotracheitis virus
S. Histomonas meleagridis

List II: Target Organs or Tissues
1. Trachea
2. Duodenum
3. Cecum and Liver
4. Joints and Tendon sheaths

(A) P-2, Q-4, R-1, S-3
(B) P-3, Q-1, R-4, S-2
(C) P-2, Q-1, R-4, S-3
(D) P-1, Q-4, R-2, S-3

2024

84. In terms of primary pathology, how does Mycoplasma synoviae visually differ from viral arthritis (reovirus) in the joints?
(A) M. synoviae induces articular urate crystal deposition, whereas viral arthritis produces extensive dry fibrinous lesions.
(B) M. synoviae causes strictly dry, non-exudative hyperplastic lesions, whereas viral arthritis induces severe suppurative arthritis.
(C) M. synoviae produces creamy to caseous yellow joint exudate, while viral arthritis exhibits clear fluid or fibrosis.
(D) M. synoviae and viral arthritis have indistinguishable gross joint lesions, meaning histographical review is strictly required.

2005

85. The presence of 'sulfur-colored' (bright yellow) feces in turkeys is a classical clinical sign indicating severe liver damage associated with:
(A) Histomoniasis
(B) Coccidiosis
(C) Fowl Cholera
(D) Turkey viral hepatitis

2008

86. Which of the following best describes the pathogenesis of ascites syndrome in broilers reared at high altitudes?
(A) Decreased barometric pressure at high altitudes causes mechanical rupturing of thin-walled pulmonary and renal capillaries.
(B) Increased systemic oxygen demands under cold stress cause left-sided ventricular valvular failure and severe pulmonary edema.
(C) Altitude-induced hypoxia directly triggers progressive hepatic cirrhosis, leading to secondary portal hypertension and ascites.
(D) Hypoxia induces pulmonary arterial vasoconstriction, leading to right ventricular hypertrophy and subsequent heart failure.

NODIA

2015

87. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R). Choose the correct option.
Assertion (A): Intestinal coccidiosis predisposes chickens to necrotic enteritis caused by Clostridium perfringens.
Reason (R): Eimeria-induced mucosal damage and plasma leakage provide a rich nutrient environment that promotes the rapid proliferation of clostridia.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2022

88. Necrotic enteritis in poultry is principally mediated by which specific exotoxin produced by Clostridium perfringens Type A or C?
(A) Botulinum neurotoxin complex
(B) Shiga-like enterotoxin
(C) Alpha toxin or NetB toxin
(D) Tetanospasmin neurotoxin

2008

89. Which of the following Eimeria species is characteristically associated with transverse white plaques ("ladder-like lesions") in the duodenum of chickens during necropsy?
(A) Eimeria tenella
(B) Eimeria acervulina
(C) Eimeria necatrix
(D) Eimeria maxima

2016

90. Which avian condition is characterized by a focal or generalized loss of feathers accompanied by severe dermal hemorrhage and gangrenous necrosis, often linked to

staphylococcal or clostridial infections following immunosuppression?
(A) Gangrenous dermatitis
(B) Erysipelas
(C) Fowl pox
(D) Squamous cell carcinoma

2025
91. Identify the incorrect combination of statements regarding avian toxicology and deficiency syndromes.
I. Encephalomalacia strictly features hemorrhage and necrosis of the cerebellum.
II. Vitamin B12 deficiency directly causes severe curled toe paralysis.
III. Polyneuritis (stargazing posture) is classic for Thiamine (B1) deficiency.
Which of the above statements are incorrect?
(A) I and II
(B) II only
(C) III only
(D) I and III

2022
92. The occurrence of "clubbed down" in unhatched embryos is pathognomonic for a deficiency in which trace nutrient?
(A) Copper
(B) Riboflavin
(C) Zinc
(D) Vitamin A

2003
93. A high incidence of blood spots within chicken eggs is often associated with a dietary deficiency of:
(A) Vitamin C
(B) Vitamin A
(C) Vitamin E
(D) Vitamin K

2017
94. The term "marble spleen disease" refers to an acute, fatal disease of pheasants. Which agent causes this disease?
(A) A lymphotropic herpesvirus
(B) An oncogenic retrovirus
(C) An aviadenovirus (Type II)
(D) A neurotropic paramyxovirus

2005
95. Identify the correct combination of statements concerning Avian Mycoplasmosis.
I. M. gallisepticum is the primary cause of Chronic Respiratory Disease (CRD) in chickens.
II. M. meleagridis exclusively affects chickens and spares turkeys.
III. Mycoplasma lacks a rigid cell wall, making penicillins ineffective for treatment.
IV. Vertical transmission through the egg is a primary route of dissemination.
Which of the above statements are correct?
(A) I, II and III
(B) I, III and IV
(C) II, III and IV
(D) I, II and IV

2018
96. "Spiking mortality syndrome" in young broilers is primarily characterized by which profound clinical chemistry abnormality just prior to death?
(A) Acute hypercalcemia
(B) Acute hypernatremia
(C) Severe hypoglycemia
(D) Acute hypokalemia

2024
97. The presence of 'tiger-stripe' lesions in the skeletal muscles (pale streaking) is histologically indicative of Zenker's necrosis. In poultry, this is highly characteristic of:
(A) Chronic aflatoxicosis inducing hepatic fatty change
(B) Ionophore toxicity (e.g., monensin overdose)
(C) Lead poisoning causing severe peripheral neuropathy
(D) Calcium deficiency leading to systemic osteomalacia

2017
98. Which heavy metal toxicity in waterfowl is classically diagnosed post-mortem by the presence of eroded gizzard mucosa, severe proventricular impaction, and green bile-stained feces?
(A) Zinc toxicity
(B) Copper toxicity
(C) Cadmium toxicity
(D) Lead toxicity

2025
99. Identify the correct combination of statements concerning avian tumor viruses.
I. The v-rel oncogene is associated with Reticuloendotheliosis virus.
II. Avian Leukosis Virus is an exogenous retrovirus.
III. Marek's disease virus replicates exclusively in the feather follicle epithelium to produce

Click Here to Buy book on Flipkart

fully infectious cell-free virus.
IV. Tumor development in ALV requires the integration of the provirus near a host cellular proto-oncogene (e.g., c-myc).
V. Marek's disease is easily eradicated because it cannot survive outside the host.

Which of the above statements are correct?
(A) I, II, III and IV
(B) I, II, IV and V
(C) II, III, IV and V
(D) I, III, IV and V

2021
100. Reticuloendotheliosis virus (REV) in chickens can cause a runting syndrome, characterized pathologically by the severe atrophy of which two organs?
(A) Brain and spinal cord parenchyma
(B) Liver and renal cortical tissue
(C) Thymus and bursa of Fabricius
(D) Proventricular wall and gizzard

2022
101. Which condition is defined as the prolapse of the oviduct, often leading to subsequent cannibalism in a flock of laying hens?
(A) "Blow-out" or vent prolapse
(B) Chronic fibrinous salpingitis
(C) Distal oviduct egg bound
(D) Internal layer syndrome

2024
102. An "egg-bound" bird has an egg lodged in the reproductive tract. This most frequently occurs in which anatomical segment?
(A) Infundibulum (proximal segment)
(B) Magnum (albumen secreting region)
(C) Isthmus (shell membrane region)
(D) Uterus (shell gland) or vagina

2004
103. Arrange the segments of the avian oviduct in the correct chronological order traversed by an ovum during egg formation.
I. Uterus
II. Infundibulum
III. Vagina
IV. Isthmus
V. Magnum

Select the correct sequence from the options below.
(A) II, V, IV, I, III
(B) II, IV, V, I, III
(C) V, II, IV, I, III
(D) II, V, I, IV, III

2007
104. In the magnum of the oviduct, what is the primary structural component added to the developing egg?
(A) The yolk
(B) The chalazae and albumen
(C) The shell membranes
(D) The calcium carbonate shell

2015
105. "White chick syndrome" is a recently characterized pathology associated with vertical transmission of which virus?
(A) Avian Astrovirus
(B) Avian Encephalomyelitis virus
(C) Avian Hepatitis E virus
(D) Chicken Astrovirus (CAstV)

2023
106. Big Liver and Spleen Disease (BLS) of broiler breeders, which causes hepatosplenomegaly and a sudden drop in egg production, is caused by:
(A) Avian adenovirus type 1
(B) Marek's disease herpesvirus
(C) Avian leukosis retrovirus
(D) Avian hepatitis E virus

NODIA

2001
107. The standard viral isolation procedure of Newcastle Disease Virus from field tissue samples involves several sequential steps:
I. Homogenization of tissues and preparation of a 10% suspension
II. Inoculation into the allantoic cavity of 9-11 day old embryonated chicken eggs
III. Collection of appropriate tissues (trachea, brain, spleen) on ice
IV. Harvesting of allantoic fluid after embryo death or after 5-7 days
V. Spot hemagglutination (HA) testing of the allantoic fluid

Which of the following represents the correct chronological sequence of these steps?
(A) III, I, II, IV, V
(B) I, III, II, IV, V
(C) III, II, I, V, IV
(D) I, II, III, V, IV

2002
108. The Mean Death Time (MDT) in embryonated chicken eggs is a standard biological index used to determine the virulence of Newcastle disease virus isolates. A lentogenic strain typically has an MDT of:
(A) Less than 60 hours
(B) 60 to 90 hours

(C) More than 90 hours
(D) Less than 24 hours

2015

109. During standard necropsy, finding a thickened, rigid, and narrowed tracheal lumen with caseous exudate in turkeys is pathognomonic for infection with:
(A) Haemophilus paragallinarum
(B) Bordetella avium
(C) Mycoplasma gallisepticum
(D) Pasteurella multocida

2013

110. Which lesion strictly distinguishes the velogenic viscerotropic form of Newcastle disease from the velogenic neurotropic form?
(A) Hemorrhagic gastrointestinal lesions
(B) Severe tracheitis and airsacculitis
(C) Non-suppurative encephalomyelitis
(D) Marked proventricular impaction

2015

111. Amyloidosis in poultry, typically a consequence of chronic antigenic stimulation (such as chronic tuberculosis or staphylococcosis), results in the deposition of amyloid primarily in which organs?
(A) Brain and spinal cord tissue
(B) Myocardium and lung parenchyma
(C) Liver, spleen, and joints
(D) Skeletal muscle bundles

2018

112. The gross appearance of a liver with amyloidosis in a bird is typically described as:
(A) Shrunken, fibrotic, and green
(B) Small, soft, and acutely hemorrhagic
(C) Completely replaced by large caseous nodules
(D) Enlarged, pale, firm, and waxy

2001

113. Fowl cholera (Pasteurellosis) may present acutely as a septicemia. The most striking and rapid vascular gross lesion observed upon opening an acute case is:
(A) Severe, dry coagulative necrosis of the spleen
(B) Diffuse, pale discoloration of the hepatic tissue
(C) Petechiae on the epicardium and abdominal fat
(D) Extensive, chronic fibrosis of the pleural air sacs

2018

114. During an outbreak of Avian Spirochaetosis (Borrelia anserina), necropsy of affected birds typically reveals an enlarged, mottled spleen. What is the characteristic finding in the liver?
(A) Severely atrophied liver with massive zones of focal necrosis
(B) Enlarged liver with necrotic foci and marginal infarcts
(C) Shrunken, fibrotic liver showing chronic cirrhotic changes
(D) Diffuse lipidosis and swelling without cellular necrosis

2021

115. A 6-week-old broiler chicken is diagnosed with pododermatitis (bumble foot). The pathogenesis typically initiates due to:
(A) Systemic bacterial spread originating from the respiratory tract
(B) Congenital skeletal malformation affecting the metatarsal bones
(C) Primary viral infection localized in the footpad epithelium
(D) Poor litter and abrasive flooring causing mechanical injury

2023

116. Curled toe paralysis results from a specific neural lesion. Histopathologically, the sciatic nerve shows:
(A) Schwann cell proliferation and myelin loss
(B) Diffuse mononuclear cell infiltration
(C) Suppurative neuritis with heterophil infiltration
(D) Focal hemorrhage with extensive perineural fibrosis

2005

117. Eimeria tenella primarily localizes and undergoes its complex life cycle, including massive schizogony, in which specific cells of the ceca?
(A) Goblet cells
(B) Villi tip enterocytes
(C) Crypt epithelial cells
(D) Lamina propria macrophages

NODIA

2006

118. Which structural form of the Eimeria parasite is responsible for penetrating the host's intestinal epithelial cells to initiate infection?
(A) Sporozoite
(B) Merozoite
(C) Oocyst
(D) Macrogamete

(B) Dark, dry breast muscle and prominent ureteral urates
(C) Massive pulmonary congestion leading to acute right-sided heart failure
(D) Disseminated petechial hemorrhages across all internal serosal surfaces

2015
137. Which lesion best differentiates acute septicemic Fowl Typhoid from Pullorum disease in adult birds?
(A) Pullorum strictly causes lung nodules, while Typhoid strictly causes joint lesions.
(B) Fowl Typhoid causes hemorrhagic ceca, while Pullorum does not affect the gut.
(C) Fowl Typhoid typically produces a distinct, swollen, bronze-green discolored liver.
(D) Pullorum disease produces severe tracheitis, whereas Typhoid does not.

2009
138. Pullorum disease and Fowl Typhoid are both caused by biovars of Salmonella enterica subsp. enterica serovar Gallinarum. Which of the following defines their motility?
(A) Highly motile by peritrichous flagella
(B) Motile via a single polar flagellum
(C) Motile via axial filaments
(D) Non-motile

2013
139. Mushy chick disease is a colloquial term for:
(A) Omphalitis
(B) Rickets
(C) Coccidiosis
(D) Avian Encephalomyelitis

2003
140. Which specific management failure during the incubation period is most likely to predispose neonates to omphalitis?
(A) Setting highly porous eggshells within a completely sanitized, clean incubator
(B) Contaminated eggshells coupled with improper incubator humidity/temperature
(C) Providing insufficient mechanical turning during the early stages of incubation
(D) Routine candling and physical handling of the delicate eggs too frequently

2025
141. The Avian paramyxovirus type 1 (APMV-1) fusion protein (F0) must be cleaved by host cellular proteases to become infective. Velogenic strains of NDV can be cleaved by:
(A) Trypsin-like proteases confined solely to respiratory and intestinal tracts
(B) Ubiquitous intracellular proteases (like furin) in most host tissues
(C) Highly specialized viral-encoded proteases active within the virion capsule
(D) Specific cellular proteases present exclusively in the central brain tissue

2005
142. Pathognomonic lesions of Infectious Coryza include acute catarrhal inflammation of the nasal passages and sinuses. Which diagnostic method confirms the causative agent, Avibacterium paragallinarum?
(A) Culturing on blood agar with a Staphylococcus nurse colony
(B) Aerobic cultivation of the sinus exudate on MacConkey agar
(C) Direct observation of microaerophilic spirochetes under dark-field
(D) Selective isolation of fungal agents on Sabouraud dextrose agar

2023
143. Which disease is characterized by a saw-horse stance, muscular spasms, and is caused by an environmental bacterium producing a neurotoxin?
(A) Avian botulism
(B) Fowl cholera
(C) Tetanus
(D) Listeriosis

2016
144. In commercial layers, a rapid and dramatic drop in egg production accompanied by the production of soft-shelled, shell-less, and depigmented eggs, without significant mortality or systemic illness, is the classic presentation of:
(A) Highly Pathogenic Avian Influenza
(B) Viscerotropic Newcastle Disease
(C) Fowl Cholera
(D) Egg Drop Syndrome (EDS-76)

2008
145. A post-mortem of a layer hen reveals a coagulated mass of yolk material in the abdominal cavity accompanied by severe fibrinous inflammation. This pathological state is clinically termed:
(A) Visceral gout
(B) Ascites
(C) Egg peritonitis
(D) Internal layer syndrome without peritonitis

Downloaded from www.pdf.tube

2025

127. Analyze the assertion and reason statements below to select the correct option.

Assertion (A): The use of unattenuated live vaccines for Infectious Laryngotracheitis (ILT) is restricted.

Reason (R): The ILT virus can establish latency in the trigeminal ganglion and recrudesce during periods of stress, potentially sparking a new outbreak.

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2017

128. Under the Avian Leukosis/Sarcoma complex, erythroblastosis refers to the intravascular proliferation of immature erythroid cells. This condition results in the classic post-mortem finding of:
(A) A shrunken, fibrotic spleen
(B) Marked pallor of all visceral organs
(C) Diffuse tumor nodules in the lung parenchyma
(D) A severely enlarged, cherry-red liver

2011

129. Avian osteopetrosis (thick leg disease) is a viral-induced skeletal pathology characterized by:
(A) Marked thinning of cortical bones predisposing the skeleton to spontaneous fractures
(B) Severe inflammatory destruction of articular cartilage resulting in complete joint fusion
(C) Diaphyseal thickening of long bones due to extreme periosteal bone formation
(D) Deposition of massive crystallized urate aggregates inside the skeletal bone marrow

2021

130. The pathogenesis of transient paralysis (classic form) associated with Marek's disease is primarily attributed to:
(A) Severe, compressive solid tumor mass formation surrounding the spinal cord
(B) Vasogenic brain edema and inflammation affecting the central nervous system
(C) Progressive nutritional depletion of vitamin E and selenium caused by the virus
(D) Physical disruption of the sciatic nerve pathways by infiltrating lymphoblasts

2025

131. Which of the following is considered an indicator of poor ventilation and high atmospheric ammonia concentrations in a commercial poultry house?
(A) Epidemic visceral gout
(B) Widespread cannibalism
(C) Fatty liver hemorrhagic syndrome
(D) Keratoconjunctivitis

2007

132. The spectacle or owl-like appearance of the eyes in poultry affected by Avian Metapneumovirus (aMPV) is primarily due to:
(A) Subcutaneous edema around the periorbital tissues
(B) Diffuse intraocular hemorrhage in both chambers
(C) Severe, irreversible bilateral lens cataracts
(D) Complete motor paralysis affecting the eyelids

2023

133. Cnemidocoptes mutans is a parasitic mite responsible for which characteristic clinical condition in poultry?
(A) Red mite infestation
(B) Depluming itch
(C) Scaly leg disease
(D) Air sac mites

NODIA

2003

134. Which class of immunoglobulins is primarily transferred from the hen to the chick via the egg yolk, providing essential systemic maternal immunity?
(A) IgY
(B) IgA
(C) IgM
(D) IgD

2001

135. The proventriculus and gizzard interface features a transition zone. Diphtheritic membranes or hemorrhagic bands strictly at this interface are a hallmark pathological finding in:
(A) Coccidiosis
(B) Fowl Typhoid
(C) Aspergillosis
(D) Newcastle Disease

2019

136. In the case of severe dehydration in poultry, necropsy typically reveals:
(A) Highly edematous, pale musculature accompanied by severe ascites

2004

174. A classic pathological finding of deep pectoral myopathy (Oregon disease) in fast-growing turkeys and broilers is:
(A) Ischemic necrosis of the supracoracoideus muscle due to compression
(B) Acute suppurative inflammation limited to the superficial pectoral muscle
(C) Severe hemorrhage coupled with complete rupture of the gastrocnemius tendon
(D) Extensive, progressive fibrosis throughout the deep thigh musculature

2022

175. During an outbreak of velogenic Newcastle disease, the characteristic intestinal lesions typically locate at:
(A) Deep necrotic ulcers within the muscularis layer of the upper duodenum
(B) Extensive hemorrhage and swelling localized to the cloacal bursa only
(C) Hemorrhagic or necrotic lesions of Peyer's patches and cecal tonsils
(D) Severe diphtheritic membranes covering the mucosal lining of the crop

2022

176. Which of the following diagnostic tests provides the most definitive confirmation and pathotyping of Highly Pathogenic Avian Influenza (HPAI)?
(A) Standard hemagglutination inhibition (HI) assays using monospecific polyclonal serves
(B) Group-specific agar gel immunodiffusion (AGID) testing of splenic tissues
(C) Detailed histopathological evaluation of the inflammatory tracheal mucosa
(D) Virus isolation followed by RT-PCR and sequencing of the HA cleavage site

2018

177. A condition known as round heart disease affects young turkeys. Pathologically, it is defined as:
(A) Idiopathic hypertrophic cardiomyopathy localized in the left ventricle
(B) Chronic, diffuse fibrinous pericarditis caused primarily by E. coli
(C) Dilated cardiomyopathy causing right or biventricular dilation
(D) Extracellular urate crystal deposition strictly on the epicardium

2014

178. What is the most common predisposing factor for the development of bumble foot (pododermatitis) in broiler flocks?
(A) Severe zinc deficiency
(B) Wet litter and poor management
(C) Calcium-phosphorus imbalance
(D) High levels of dietary protein

2010

179. The physiological cause of tibial dyschondroplasia in broilers is best described as:
(A) Excessive, rapid osteoclast-mediated resorption of cortical bone without compensatory synthesis
(B) Severe, acute inflammatory destruction of the chondrocytes within the epiphyseal plate
(C) A specific congenital defect involving structurally abnormal collagen matrix synthesis
(D) Failure of mature cartilage to calcify and undergo vascular invasion, leaving a retained plug

2019

180. A definitive gross diagnosis of syngamiasis (gapeworm infection) can be made by finding the characteristic Y-shaped adult worms in the:
(A) Trachea
(B) Crop
(C) Small intestine
(D) Ceca

2004

181. In an avian necropsy, finding a grossly enlarged, mottled liver with pinpoint white necrotic foci, combined with a massively enlarged spleen, in an adult chicken is highly suggestive of:
(A) Fowl Cholera
(B) Marek's Disease
(C) Fowl Typhoid
(D) Avian Tuberculosis

2005

182. A flat, non-elevated, pale area on the comb of a bird recovering from an infection most likely represents the healed phase of:
(A) Dry pox (Fowl Pox)
(B) Favus (Dermatophytosis)
(C) Swollen head syndrome
(D) Erysipelas

2005

183. Which of the following describes the pathogenesis of early chick mortality due to yolk sac infection?
(A) Ascending bacterial migration originating directly from the cloacal opening post-hatching
(B) Localized infection of the anatomical umbilicus that remains confined without systemic spread

Click Here to Buy book on Amazon

2023
146. Which of the following definitions best describes Cage Layer Fatigue?
(A) Generalized muscle atrophy resulting from a complete lack of exercise in small cages
(B) Severe metabolic osteodystrophy causing paralysis in high-yielding caged hens
(C) Intense psychological stress within crowded environments leading directly to cannibalism
(D) Acute peripheral circulatory failure resulting from prolonged and continuous standing

2008
147. A bird presenting with dyspnea, gasping, and expectoration of bloody mucus typically suffers from an acute episode of:
(A) Infectious Bronchitis
(B) Avian Encephalomyelitis
(C) Mycoplasma gallisepticum
(D) Infectious Laryngotracheitis

2011
148. Differentiating between Marek's disease (MD) and Avian Leukosis (ALV) based on age of onset is a useful clinical guide. Which statement is generally correct?
(A) MD peaks around 12-24 weeks; ALV rarely occurs before 14-16 weeks.
(B) MD occurs strictly under 4 weeks; ALV peaks between 4 and 8 weeks.
(C) Both viral conditions manifest only after birds reach sexual maturity.
(D) ALV affects neonatal chicks, while MD affects only fully mature adults.

2009
149. Biliary hyperplasia and periportal fibrosis in a bird's liver are chronic histological indicators primarily associated with ingestion of:
(A) Ochratoxin A
(B) Zearalenone
(C) Aflatoxin
(D) Monensin

2019
150. Renal gout occurs when the kidney's excretory capacity is exceeded. The avian kidney lacks which crucial mammalian structure, making it highly dependent on tubular secretion for uric acid clearance?
(A) Loops of Henle
(B) Functional glomeruli
(C) Proximal tubules
(D) Functional ureters

NODIA

2019
151. Hypoglycemia in birds affected by Fatty Liver and Kidney Syndrome (FLKS) is thought to arise primarily from a failure in gluconeogenesis due to the deficiency of which biotin-dependent enzyme?
(A) Hexokinase
(B) Phosphofructokinase
(C) Glycogen synthase
(D) Pyruvate carboxylase

2012
152. Infectious Stunting Syndrome (Malabsorption Syndrome) typically affects broiler chicks. Which of the following gross features is NOT characteristic of this syndrome?
(A) Distended, pale intestines with undigested feed
(B) Massive hypertrophy of the bursa of Fabricius
(C) Pale, small pancreas
(D) Retention of embryonic feathers and stunting

2009
153. A layer flock is experiencing a slow increase in mortality. Necropsy reveals tumor masses in the liver, spleen, and bursa. Histopathology shows a uniform population of lymphoblasts originating in the bursa. This defines:
(A) Marek's disease
(B) Reticuloendotheliosis
(C) Lymphoid leukosis
(D) Inclusion body hepatitis

2005
154. Fowl tick fever is an alternate name for which avian disease due to its transmission by Argas persicus?
(A) Avian malaria
(B) Fowl cholera
(C) Infectious coryza
(D) Avian spirochaetosis

2013
155. A hallmark of severe Infectious Bursal Disease (Gumboro) is the profound depletion of B-lymphocytes in the bursa. The long-term consequence of early IBDV infection (before 2 weeks of age) is:
(A) Permanent immunosuppression from destruction of precursor B-cells
(B) Temporary immunosuppression that rebounds completely within several weeks
(C) Highly enhanced T-cell mediated cellular immunity acting to compensate
(D) Complete autoimmune destruction affecting

2009

119. A condition known as crop impaction or sour crop in poultry is a functional stasis often secondary to:
(A) Standard diets completely lacking in insoluble grit
(B) Excessive levels of highly concentrated dietary protein
(C) Sudden, uncontrolled overconsumption of drinking water
(D) Ingestion of tough grasses or foreign bodies

2011

120. Avian Tuberculosis, caused by Mycobacterium avium subsp. avium, forms classic granulomas in the liver and spleen. These granulomas are distinctively characterized by:
(A) Central zone of massive dystrophic calcification surrounded by neutrophils
(B) Central caseous necrosis without calcification, surrounded by giant cells
(C) Purely diffuse lymphocytic infiltrates showing no cellular or tissue necrosis
(D) Intense eosinophilic infiltrates closely resembling localized allergic reactions

2011

121. The most prevalent natural route of infection for Mycobacterium avium in commercial and backyard poultry is:
(A) Direct inhalation of infectious aerosols
(B) Vector-borne transmission via mosquitoes
(C) Ingestion of contaminated soil or water
(D) Vertical transmission directly via the egg

2024

122. During the histological evaluation of an avian lung infected with Aspergillus fumigatus, the characteristic fungal hyphae observed using a Gomori Methenamine Silver (GMS) stain are:
(A) Broad, non-septate, and branching at right angles
(B) Elongated pseudohyphae with prominent budding yeast forms
(C) Small, intracellular yeast forms entirely lacking hyphae
(D) Narrow, septate, and branching at acute angles

2017

123. Match the toxins in List I with their primary pathological targets or classic clinical signs in poultry in List II.
List I: (Toxins)
P. Botulinum toxin
Q. Aflatoxin B1
R. T-2 toxin (Trichothecene)
S. Monensin (overdose)
List II: (Pathological Targets or Clinical Signs)
1. Oral lesions and immunosuppression
2. Hepatocellular necrosis and carcinoma
3. Flaccid paralysis (Limberneck)
4. Skeletal and cardiac muscle necrosis
(A) P-3, Q-2, R-1, S-4
(B) P-2, Q-3, R-1, S-4
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-2, S-3

2002

124. Limberneck in waterfowl and chickens is a fatal paralytic disease caused by the ingestion of the pre-formed toxin of Clostridium botulinum. This toxin exerts its effect by:
(A) Causing massive, progressive demyelination of the spinal cord
(B) Blocking acetylcholine release at the neuromuscular junction
(C) Inducing continuous release of glycine, causing spastic paralysis
(D) Destroying the functional motor cortex region of the brain

2023

125. Which characteristic of the avian respiratory system facilitates the rapid systemic spread of inhaled pathogens like aerosolized highly pathogenic avian influenza?
(A) A rigid fibromuscular diaphragm that mechanically forces air into blood vessels
(B) Unidirectional airflow and thin-walled air sacs contacting visceral organs
(C) A highly ciliated, functional pseudo-alveolar system identical to mammals
(D) Complete anatomic separation of the upper and lower respiratory tracts

2009

126. A distinctive pathological feature of Avian Infectious Bronchitis Virus (IBV) nephropathogenic strains is the gross presentation of the kidneys, which appear:
(A) Pale, swollen, and mottled with urate-distended tubules
(B) Severely atrophied, shrunken, and dark black-green
(C) Small, abnormally firm, and filled with multiple cysts
(D) Moderately hemorrhagic with massive focal infarctions

Downloaded from www.pdf.tube

(B) Hypovitaminosis A
(C) Wet pox
(D) Trichomoniasis

2008

166. A critical difference between the plaques of Wet Pox and the lesions of Vitamin A deficiency in the esophagus is:
(A) Vitamin A lesions are expressible pustules, while pox lesions are firmly attached fibrinous plaques.
(B) Wet pox lesions can be easily scraped off without damage, while Vitamin A lesions are deeply embedded.
(C) Wet pox causes strictly hemorrhagic mucosal ulcerations, while Vitamin A deficiency causes dry black scabs.
(D) There is no visible, reliable gross pathological difference between the two disease manifestations.

2014

167. Analyze the assertion and reason statements below to select the correct option.
Assertion (A): Hemorrhagic enteritis of turkeys, caused by an aviadenovirus, often leads to sudden death characterized by bloody droppings.
Reason (R): The virus replicates in the intestinal epithelium, directly destroying the mucosal lining and causing massive capillary hemorrhage.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2017

168. Which of the following viruses is known to cause a drop in egg production characterized by misshapen, rough, and chalky eggs without the severe systemic illness seen in Newcastle disease or Avian Influenza?
(A) Avian Influenza (low path)
(B) Infectious Bronchitis Virus
(C) Avian Encephalomyelitis Virus
(D) Fowlpox Virus

2006

169. What is the fundamental pathological change in the pathogenesis of Fatty Liver Haemorrhagic Syndrome (FLHS)?
(A) Massive infiltration of inflammatory heterophils throughout the liver parenchyma
(B) Severe, acute coagulative necrosis of hepatocytes directly caused by mycotoxins
(C) Hepatocellular triglyceride accumulation leading to capsular rupture and hemorrhage
(D) Direct viral lysis of hepatic cell membranes causing acute, localized bleeding

2006

170. Which of the following conditions represents an important zoonotic occupational hazard for personnel performing necropsies on birds suffering from severe fibrinous airsacculitis, hepatomegaly, and splenomegaly?
(A) Marek's disease
(B) Infectious bursal disease
(C) Avian leukosis
(D) Chlamydiosis

2009

171. A characteristic gross lesion of Mycoplasma synoviae infection in the acute phase involves:
(A) Creamy, viscous exudate in the keel bursa and joint spaces
(B) Marked enlargement and purulent exudate limited to the footpad
(C) Massive, acute hemorrhage directly into the surrounding articular space
(D) Severe destruction of articular cartilage leading to chronic joint fusion

2021

172. The presence of large numbers of heterophils in an avian inflammatory lesion is equivalent to the presence of which cell type in a mammalian lesion?
(A) Macrophages
(B) Neutrophils
(C) Lymphocytes
(D) Eosinophils

2013

173. Which of the following is true regarding avian heterophils compared to mammalian neutrophils?
(A) Avian heterophils possess high concentrations of active myeloperoxidase comparable to mammalian cells.
(B) Avian heterophils lack significant myeloperoxidase, relying on non-oxidative mechanisms for killing.
(C) Avian heterophils function as the primary cellular defense against systemic viral rather than bacterial agents.
(D) Avian heterophils are structurally classified as mononuclear phagocytes rather than granulocytes.

NODIA

the functional thyroid gland

2011

156. In diagnosing Gumboro disease via gross pathology, the presence of hemorrhages at the juncture of the proventriculus and gizzard can resemble Newcastle disease. However, IBD usually uniquely presents with:
(A) Simultaneous development of severe tracheitis
(B) Pectoral and thigh muscle hemorrhages
(C) Multifocal tumor nodules throughout the spleen
(D) Pinpoint petechial lesions on the comb and wattles

2009

157. Which viral disease primarily utilizes the respiratory route for infection but establishes a lifelong latent infection in the trigeminal ganglion?
(A) Newcastle disease virus
(B) Infectious laryngotracheitis virus
(C) Infectious bronchitis virus
(D) Marek's disease herpesvirus infection

2015

158. Pathognomonic focal necrosis of the liver with corresponding ceca core formation (necrotic typhlitis) in peafowl and turkeys is diagnostic for:
(A) Histomoniasis
(B) Coccidiosis
(C) Salmonellosis
(D) Pasteurellosis

2010

159. The clinical sign of torticollis (wry neck) in poultry can be caused by all of the following EXCEPT:
(A) Newcastle disease
(B) Vitamin E deficiency (Encephalomalacia)
(C) Biotin deficiency
(D) Pasteurella multocida (localized cranial infection)

2024

160. During post-mortem, an inspector finds a solid, pale, firm tumor mass enveloping the ovary of a 20-week-old pullet, with similar firm, gray-white nodules in the heart and lungs. The sciatic nerves are thickened. This is indicative of:
(A) Ovarian adenocarcinoma
(B) Avian Leukosis
(C) Egg bound condition
(D) Marek's Disease

2009

161. A distinguishing histological feature of Marek's disease compared to Lymphoid leukosis is:
(A) A strictly uniform cellular population consisting entirely of large lymphoblasts
(B) Presence of pathognomonic, prominent eosinophilic intracytoplasmic inclusions
(C) A pleomorphic neoplastic infiltrate of lymphocytes, plasma cells, and macrophages
(D) Complete histopathological absence of any inflammatory or reactive host cells

2003

162. Which avian respiratory disease agent targets the ciliated epithelium of the trachea, causing deciliation and predisposing the bird to secondary E. coli infections (the classic CRD complex)?
(A) Avian Pneumovirus
(B) Mycoplasma gallisepticum
(C) Infectious Laryngotracheitis virus
(D) Fowlpox virus

2013

163. Match the metabolic/nutritional conditions in List I with their primary causative factors in List II.
List I: (Metabolic or Nutritional Conditions)
P. Perosis
Q. Ascites syndrome
R. Exudative diathesis
S. Visceral gout
List II: (Causative Deficiencies or Excesses)
1. Vitamin E / Selenium deficiency
2. Manganese / Choline deficiency
3. High protein/calcium diet coupled with water deprivation
4. Hypoxia and rapid growth
(A) P-2, Q-4, R-1, S-3
(B) P-4, Q-2, R-1, S-3
(C) P-2, Q-1, R-4, S-3
(D) P-1, Q-2, R-3, S-4

2008

164. The principal cause of hydropericardium syndrome (Angara disease) in broilers is:
(A) Fowl adenovirus serotype 4
(B) Avian reovirus
(C) Infectious bronchitis virus
(D) Fowlpox virus

2010

165. The presence of discrete, elevated, white-to-yellow plaques in the oral cavity, esophagus, and crop that are difficult to peel away without causing bleeding is characteristic of:
(A) Candidiasis

Click Here to Buy book on Flipkart

(C) Co-infection with specific immunosuppressive viral agents required for disease manifestation
(D) Bacterial entry through the unhealed navel into the yolk sac, causing systemic septicemia

2002

184. The bursa of Fabricius attains its maximum size relative to body weight at approximately what age in the chicken, before beginning physiological involution?
(A) 1-2 weeks
(B) 3-8 weeks
(C) 12-16 weeks
(D) 20-24 weeks

2022

185. In Avian Mycoplasmosis caused by Mycoplasma gallisepticum, which of the following is NOT a typical presentation?
(A) Catarrhal exudate in the nasal passages
(B) Fibrinous airsacculitis when complicated by E. coli
(C) Severe hemorrhagic tracheitis with blood clots
(D) Mild conjunctivitis and facial edema

2003

186. Which mycotoxin is a potent immunosuppressant and primarily recognized by oral and gastrointestinal necrotic lesions, severely impacting the bird's performance?
(A) Aflatoxin B1
(B) Fumonisin B1
(C) Ergotamine
(D) T-2 toxin

2009

187. The term brooder pneumonia is synonymous with an acute respiratory outbreak in young chicks caused by:
(A) Aspergillus fumigatus
(B) Avian metapneumovirus
(C) Infectious bronchitis virus
(D) Mycoplasma gallisepticum

2019

188. In cases of acute lead poisoning in waterfowl, which of the following neurological signs is a classic diagnostic feature before death?
(A) Torticollis and backward circling
(B) Wing droop and leg paresis
(C) Clonic-tonic convulsions
(D) Blindness and stargazing

2001

189. A distinguishing feature of a normal, active avian ovary in a laying hen compared to a non-layer is the presence of:
(A) A single, small, solid cluster of pale ova
(B) A hierarchy of large, yolk-filled follicles
(C) Multiple large cystic follicles containing clear fluid
(D) A uniform mass of caseous material

2008

190. Histomoniasis typically causes extensive damage to the ceca and liver. The transmission of Histomonas meleagridis relies on the egg of Heterakis gallinarum. What role does the earthworm play in this cycle?
(A) It acts as a paratenic host, harboring infected Heterakis eggs in its tissues.
(B) It functions as the obligate definitive host for sexual replication of Histomonas.
(C) It provides specific, essential growth factors needed for the protozoan to replicate.
(D) It directly infects hepatic parenchymal tissues when physically ingested by turkeys.

2019

191. Coccidiosis in poultry is managed strictly with anticoccidials or vaccination. The choice of Eimeria strains in a live vaccine depends on ensuring coverage of:
(A) Pathogenic Eimeria species derived from diverse mammalian hosts
(B) Only Eimeria tenella, as it provides cross-immunity to all others
(C) Multiple species of Eimeria as immunity is species-specific
(D) Inactivated Eimeria oocysts to prevent any subclinical disease

2016

192. Which Eimeria species uniquely causes pathological lesions (white plaques or petechiae) in the upper half of the small intestine (duodenum)?
(A) Eimeria brunetti
(B) Eimeria tenella
(C) Eimeria necatrix
(D) Eimeria acervulina

2014

193. Egg Peritonitis is a major cause of mortality in laying flocks. The bacterial pathogen most frequently isolated from the caseous exudate in the abdominal cavity is:
(A) Salmonella Enteritidis
(B) Pasteurella multocida

Downloaded from www.pdf.tube

61. (A) Respiratory alkalosis
Panting in heat-stressed poultry is an active cooling mechanism that increases respiratory rate to dissipate heat. This hyperventilation causes excessive loss of carbon dioxide from the lungs. The reduction in blood carbon dioxide levels disrupts the carbonic acid-bicarbonate buffer system, leading to an increase in blood pH, which results in respiratory alkalosis.

62. (C) Reduction in blood bicarbonate levels due to respiratory alkalosis
Severe heat stress in laying hens induces hyperventilation, which leads to respiratory alkalosis and a decrease in blood carbon dioxide levels. This reduction limits the availability of bicarbonate ions in the shell gland. Bicarbonate is essential for calcium carbonate shell formation, and its depletion directly results in thin-shelled or shell-less eggs.

63. (D) Pancreas
Blue comb, also known as pullet disease or avian monocytosis, is a metabolic and infectious syndrome in chickens and turkeys. It is characterized by severe dehydration, cyanosis of the comb and wattles, and hemoconcentration. Post-mortem examination reveals diagnostic focal areas of necrosis in the pancreas, alongside acute renal changes and skeletal muscle dehydration.

64. (C) Broad Breasted Bronze Turkeys
A pendulous crop is a condition characterized by a dilated, flaccid crop that fails to empty, leading to fluid accumulation and mucosal inflammation. While it can be triggered by dietary factors or infections, a strong genetic predisposition for this anatomical defect is well-documented in Broad Breasted Bronze Turkeys, particularly when exposed to hot weather.

65. (B) Avian Metapneumovirus
Swollen head syndrome is an infectious respiratory complex in poultry. It is initiated by Avian Metapneumovirus, which causes inflammation of the upper respiratory tract. This viral infection is subsequently complicated by secondary bacterial pathogens, primarily Escherichia coli, leading to severe cellulitis and purulent exudate in the subcutaneous tissues of the head and face.

66. (D) Keel bone (sternum) of the breast
Breast blisters, or sternal bursitis, are inflammatory lesions that develop on the breast of heavy meat birds. They occur due to chronic pressure, friction, and trauma to the skin over the keel bone when birds spend extended periods resting on wet or abrasive litter, resulting in fluid accumulation in the subcutaneous bursa.

67. (A) Staphylococcus aureus
Bumble foot, or plantar pododermatitis, is a chronic, localized inflammatory condition of the footpad in poultry. It begins with microtrauma or skin abrasions that allow bacterial entry. Staphylococcus aureus is the most common pathogen isolated from these lesions, causing purulent and granulomatous inflammation that can extend to deeper tendons and bones.

68. (C) Sodium and amino acid (methionine) deficiency
Cannibalism in poultry flocks is a destructive behavioral vice that is often triggered by environmental or nutritional imbalances. Inadequate dietary levels of sodium or essential sulfur amino acids, particularly methionine, can cause birds to seek these nutrients. This deficiency leads to increased feather pecking and aggressive behavior towards flock mates.

NODIA

69. (A) Hemagglutinin (HA)
The virulence of avian influenza viruses is determined by the structure of the hemagglutinin surface glycoprotein. Highly Pathogenic Avian Influenza viruses possess a sequence of multiple basic amino acids at their proteolytic cleavage site. This allows the hemagglutinin to be cleaved by ubiquitous host proteases, enabling systemic viral replication in multiple organs.

70. (D) Between the proventriculus and gizzard
Highly Pathogenic Avian Influenza causes severe systemic vascular damage in poultry. In addition to facial edema and cyanosis of the comb and wattles, necropsy of affected chickens classically reveals striking band-like hemorrhages in the mucosa of the proventriculus, particularly at the junction between the proventriculus and the gizzard, reflecting widespread viral-induced vascular necrosis.

71. (B) Subcutaneous edema of the head and neck
While Newcastle disease and Highly Pathogenic Avian Influenza share many clinical and

(C) Magnum segment
(D) Vaginal sphincter

2004

204. Blackhead disease derives its name from the cyanotic discoloration of the head. However, this clinical sign is:
(A) Present in 100% of cases and pathognomonic
(B) Highly variable and not always present, despite the disease name
(C) Caused strictly by a secondary bacterial infection
(D) A direct result of localized protozoal replication in the brain

2015

205. The primary organ responsible for the generation of T-lymphocytes in the avian immune system is the:
(A) Bursa of Fabricius
(B) Thymus
(C) Spleen
(D) Bone marrow

2010

206. Avian influenza viruses are classified by subtypes based on their surface antigens. The subtypes most frequently associated with highly pathogenic strains in poultry are:
(A) H5 and H7
(B) H1 and H3
(C) H9 and H10
(D) H2 and H4

2011

207. A characteristic gross lesion of systemic acute Erysipelas (Erysipelothrix rhusiopathiae) in turkeys is:
(A) Massive, firm, solid white tumor masses located within the bursa
(B) Severe ascites accompanied by marked round heart manifestations
(C) Swollen, purplish snood and intramuscular hemorrhages
(D) Curled toe paralysis and widespread peripheral neuritis changes

2023

208. When performing a routine necropsy on a chicken, the cecal tonsils are examined. These are anatomically located at the:
(A) Distal end (blind tip) of the ceca
(B) Mid-point of the jejunum region
(C) Junction of proventriculus and gizzard
(D) Proximal end near the ileocecal junction

2001

209. The presence of green-colored, gelatinous material under the skin (subcutaneous edema) and liver necrosis in a dead chicken suggests an acute bacterial septicemia strongly associated with:
(A) Staphylococcus aureus
(B) Salmonella Pullorum
(C) Pseudomonas aeruginosa
(D) Clostridium colinum

2013

210. Which viral respiratory pathogen of poultry specifically induces the formation of syncytia (multinucleated giant cells) in the tracheal epithelium as part of its characteristic cytopathology?
(A) Newcastle disease virus
(B) Infectious laryngotracheitis
(C) Avian influenza virus
(D) Infectious bronchitis virus

****

Click Here to Buy book on Amazon

20. (C) Hydropericardium syndrome
Hydropericardium syndrome is an acute infectious disease caused by fowl adenovirus serotype four, primarily affecting young broilers. The hallmark necropsy finding is the accumulation of up to ten milliliters of clear, straw-colored fluid in the pericardial sac. This cardiac compression causes acute pulmonary congestion and liver necrosis, resulting in sudden flock mortality.

21. (A) Waterfowl
Egg Drop Syndrome-76 is caused by an atadenovirus that has a natural evolutionary reservoir in waterfowl, such as ducks and geese. While the virus can cause severe egg production drops in commercial laying chickens, these wild and domestic waterfowl species remain the primary natural hosts, carrying the virus asymptomatically and shedding it into the environment.

22. (D) Basophilic or eosinophilic intranuclear inclusion bodies
Inclusion body hepatitis is caused by fowl adenoviruses and primarily targets hepatocytes in young broilers. The diagnostic feature observed on histopathological examination of the liver is the presence of prominent intranuclear inclusion bodies within hepatocytes. These inclusions may be basophilic or eosinophilic, representing active areas of viral macromolecular synthesis and assembly.

23. (A) III, I, V, II, IV
The pathogenesis of avian reovirus-induced tenosynovitis begins with oral-fecal exposure to the virus. Initial viral replication occurs in the enteric tract and bursa, followed by a transient viremia that disseminates the virus systemically. This leads to severe inflammation of the gastrocnemius tendon sheath, eventually causing tissue fibrosis and catastrophic rupture of the tendon.

24. (C) Proventricular enlargement with pale, flaccid walls
Malabsorption syndrome, frequently linked to avian reovirus infections in young chicks, alters the upper digestive tract. The disease causes a characteristic enlargement of the proventriculus, which develops pale, flaccid walls. This structural alteration compromises gastric motility and enzyme secretion, leading to poorly digested feed, stunted growth, and poor flock uniformity.

25. (D) Misshapen, discolored, and pedunculated ovarian follicles
In adult hens, chronic infection with Salmonella Pullorum is typically localized within the reproductive system. The hallmark post-mortem finding in carrier birds consists of misshapen, discolored, cystic, and pedunculated ovarian follicles containing oily or caseous material. This chronic localization leads to reduced egg production, decreased fertility, and persistent vertical transmission of the pathogen.

26. (A) Salmonella Gallinarum
Fowl Typhoid is a severe systemic bacterial disease caused by Salmonella Gallinarum, primarily affecting mature poultry. Affected birds develop acute or chronic septicemia, with the liver becoming markedly enlarged, congested, and classically exhibiting a bronze or greenish-bronze discoloration. This color change results from pigment accumulation and focal necrosis within the hepatic parenchyma.

27. (A) III, I, IV, II
Systemic colibacillosis in poultry begins with the inhalation of dust contaminated with avian pathogenic Escherichia coli. The bacteria colonize the respiratory tract, leading to localized airsacculitis. The pathogens then cross the mucosal barrier into the bloodstream, causing septicemia and culminating in classic fibrinous polyserositis, including pericarditis and perihepatitis.

NODIA

28. (B) Caseous/Cheesy
In chronic respiratory disease caused by Mycoplasma gallisepticum, the inflammatory response in the air sacs undergoes structural progression. The initial serous or mucoid exudate becomes increasingly cellular as heterophils accumulate. Over time, the exudate dehydrates and consolidates, transforming into a dense, yellow, caseous or cheesy mass that occupies the air sac cavities.

29. (A) The wattles
Chronic fowl cholera, caused by localized infections of Pasteurella multocida, is characterized by persistent inflammatory lesions. In chickens, the wattles are a classic anatomical site for this localized form, where infection leads to unilateral or bilateral swelling, edema, and accumulation of caseous exudate. This condition is commonly referred to as swollen wattle disease.

hemorrhagic lesions throughout the digestive tract, resulting in rapid flock mortality.

10. (A) Hemagglutinin-neuraminidase (HN) protein
The hemagglutinin-neuraminidase glycoprotein is a critical surface protein located on the envelope of the Newcastle disease virus. This multifunctional protein mediates viral attachment to sialic acid receptors on host cell membranes, facilitating the initial steps of infection. It works in close conjunction with the fusion protein to allow viral entry into target cells.

11. (B) II only
Infectious bronchitis virus is a coronavirus that exhibits tropism for multiple epithelial surfaces. It causes severe respiratory disease and also directly targets the reproductive tract, leading to permanent damage to the oviduct in young chicks and hens. Therefore, claiming that this virus affects only the lower respiratory tract without inducing reproductive issues is incorrect.

12. (B) Intranuclear inclusion bodies (Cowdry type A)
Diagnostic confirmation of infectious laryngotracheitis relies on identifying specific microscopic changes within the tracheal epithelium. The presence of intranuclear, acidophilic Cowdry type A inclusion bodies inside multinucleated syncytial cells is pathognomonic for this herpesviral infection. These structures represent active sites of viral replication and assembly during the acute phase of the disease.

13. (A) Infectious bronchitis
Infectious bronchitis virus causes significant pathology in the reproductive tract of laying hens. The virus replicates in the epithelial cells of the magnum and uterus, disrupting the secretion of albumen and shell formation. This tissue damage results in laying thin, watery, misshapen, or wrinkled eggs, causing severe economic losses in commercial poultry operations.

14. (D) Score 4
Severe damage to the bursa of Fabricius in infectious bursal disease is quantified using a standardized grading scale. A score of four is assigned to a bursa that exhibits profound atrophy, shrinking to the size of a small seed. This advanced stage represents near-complete lymphoid depletion and fibroplasia following the acute inflammatory phase of infection.

15. (A) I and III
Infectious bursal disease virus primarily targets and replicates within actively dividing IgM-bearing B lymphocytes in the bursa of Fabricius rather than hepatic macrophages. Furthermore, highly virulent strains of the virus cause extremely high mortality and lead to severe, widespread depletion of these target immune cells, rendering the specified claims regarding pathogenesis incorrect.

16. (C) Gangrenous dermatitis
Subclinical Gumboro disease causes severe immunosuppression by destroying B lymphocytes in young chicks. This compromised immune state predisposes broiler flocks to opportunistic bacterial infections. Gangrenous dermatitis, caused by clostridial or staphylococcal species, is a common secondary disease that becomes highly active and lethal in these immunologically defenseless birds.

17. (B) Peripheral nerves
Marek's disease and avian leukosis are both neoplastic conditions of poultry, but they exhibit distinct pathological profiles. The frequent involvement of peripheral nerves, such as the sciatic and vagus nerves, is a key diagnostic feature of Marek's disease. These nerves appear swollen and lose their striations, which is absent in cases of avian leukosis.

18. (D) Lymphoblasts and uniformly large B-cells
Neoplastic lesions induced by avian leukosis virus consist of a homogeneous population of lymphoid cells. Microscopic evaluation of these tumors reveals uniformly large B-lymphocytes and lymphoblasts, representing monoclonal proliferation originating from the bursa of Fabricius. This cellular uniformity contrasts with the pleomorphic lymphoid populations typically seen in Marek's disease lesions.

19. (A) I, II and IV
Marek's disease is caused by Gallid alphaherpesvirus 2 and commonly presents as ocular lymphomatosis in chickens. In contrast, avian leukosis typically manifests as nodular visceral tumors in birds older than fourteen weeks. Marek's disease tumors consist of T-cells rather than B-cells, and the virus is not vertically transmitted through the egg.

Downloaded from www.pdf.tube

ANSWERS

1. (B) Mid-ventral line from the intermandibular space to the cloaca
Standard avian necropsy protocols mandate initiating the primary incision along the mid-ventral line, extending from the intermandibular space to the cloaca. This specific pathway offers optimal anatomical exposure of the visceral organs while minimizing damage to superficial tissues. It facilitates a systematic examination of the gastrointestinal, respiratory, and reproductive systems in a highly structured manner.

2. (C) 1:10 tissue to fixative ratio
Adequate preservation of avian tissues for histological examination requires a standard volumetric ratio of one part tissue to ten parts fixative. This specific proportion ensures rapid and complete penetration of the formalin into the cellular structures, preventing post-mortem autolysis. Insufficient volumes of fixative compromise cellular morphology and hinder accurate diagnostic assessment during microscopic evaluation.

3. (A) P-2, Q-1, R-4, S-3
Avian lymphoid organs possess unique anatomical and functional distributions essential for immune defenses. The bursa of Fabricius serves as the primary site for B-cell maturation, while the thymus is a lobulated organ along the vagus nerve. The spleen acts as a secondary lymphoid organ lacking distinct zones, and the Harderian gland is a paraocular plasma cell aggregation.

4. (B) Acute phase (day 3-4 post-infection)
Infectious bursal disease virus targets the bursa of Fabricius, causing severe inflammatory changes. During the acute phase of infection, typically occurring three to four days post-exposure, the bursa becomes profoundly enlarged, edematous, hyperemic, and covered with a distinctive yellowish gelatinous transudate. This characteristic presentation serves as a key diagnostic indicator of active viral replication in young chickens.

5. (A) IgA
The Harderian gland is a critical paraocular lymphoid organ in poultry that contains a dense population of plasma cells. It is primarily responsible for the local synthesis and secretion of secretory immunoglobulin A. This antibody class provides essential mucosal immunity, protecting the ocular surface and upper respiratory tract against invading pathogenic organisms in avian species.

NODIA

6. (B) I and III
Omphalitis represents a non-contagious inflammatory condition of the navel, frequently caused by opportunistic bacteria entering through unhealed tissues. This pathology, often referred to as navel ill, is commonly linked to poor hatchery sanitation, high humidity during incubation, or improper temperature management, whereas yolk sac infection is not exclusively caused by vertical transmission from breeder hens.

7. (C) A is true but R is false.
Chicks suffering from omphalitis frequently exhibit retained or unabsorbed yolk sacs due to localized inflammatory processes and systemic compromise. However, bacterial proliferation in the umbilicus produces harmful toxins that actively impair, rather than accelerate, the physiological absorption of the yolk. Consequently, the assertion is correct, but the accompanying reason is scientifically inaccurate.

8. (C) Escherichia coli
Pathogenic strains of Escherichia coli are the most common bacterial pathogens isolated from cases of coliform septicemia in neonatal chicks. This systemic infection often enters through an unhealed navel, leading to inflammation and preventing the normal absorption of the yolk sac. Affected chicks show high mortality within the first week of life.

9. (D) Velogenic viscerotropic pathotype
Pathognomonic pinpoint hemorrhages at the tips of the proventricular glands are a hallmark gross lesion of Newcastle disease. This lesions is highly characteristic of the velogenic viscerotropic pathotype, which is the highly virulent, systemic form of the virus. This pathotype causes severe

30. (C) Leishman's or Methylene blue stain
Demonstrating the characteristic bipolar morphology of Pasteurella multocida is crucial for diagnosing acute fowl cholera. Staining a blood smear or tissue impression smear with Leishman's or methylene blue stain clearly reveals these Gram-negative coccobacilli, which show concentrated staining at both cellular poles, resembling safety pins, which is diagnostic for this organism.

31. (D) Fibrinous serositis (pericarditis and airsacculitis)
Avian chlamydiosis, a zoonotic disease caused by Chlamydia psittaci, leads to systemic pathology in birds. Post-mortem examination classically reveals severe fibrinous serositis, characterized by thick deposits of yellow fibrin covering the pericardium, air sacs, and liver capsule. Affected birds also exhibit marked splenomegaly and hepatomegaly with multifocal areas of parenchymal necrosis.

32. (C) Argas persicus (fowl tick)
Avian spirochaetosis is a severe septicemic disease of poultry caused by the spirochete Borrelia anserina. This pathogen is primarily transmitted through the bite of the fowl tick, Argas persicus, which serves as the principal biological vector. The disease is characterized by high fever, depression, cyanosis, and marked enlargement of the spleen.

33. (B) Yellow-white caseous nodules in the lungs and air sacs
Aspergillosis, commonly referred to as brooder pneumonia in young chicks, results from inhaling fungal spores. The disease is characterized by the formation of multiple yellow-white, firm, caseous nodules throughout the lung parenchyma and air sac membranes. Histological examination reveals fungal hyphae surrounded by granulomatous inflammatory infiltrates, which is highly diagnostic.

34. (D) Candida albicans
Crop mycosis, or thrush, is an opportunistic fungal infection of the upper digestive tract primarily caused by the yeast Candida albicans. It is associated with prolonged antibiotic therapy or poor sanitation. The disease produces circular, white, raised, pseudomembranous patches that coalesce, creating a characteristic wrinkled, "terry-towel" appearance of the crop mucosa.

35. (A) Fatty change, biliary hyperplasia, and fibrosis
Chronic ingestion of sublethal levels of aflatoxins leads to severe, progressive hepatotoxicity in poultry. The liver becomes pale, firm, and shrunken due to widespread lipid accumulation within hepatocytes. Microscopically, this is characterized by severe fatty change, marked proliferation of bile duct epithelial cells, and extensive periportal fibrosis, which compromises hepatic function.

36. (C) Renal system
Ochratoxin A is a highly potent mycotoxin that targets the renal system in poultry. It causes severe nephrotoxicity, leading to swollen, pale kidneys with prominent urate deposits in the tubules. This renal impairment disrupts normal excretion, resulting in visceral gout and high mortality due to secondary uremia in affected avian flocks.

37. (A) Oral and proventricular mucosal necrosis (oral lesions)
Ingestion of trichothecene mycotoxins, such as T-2 toxin, causes severe contact necrosis of mucosal surfaces in poultry. Affected birds classically present with yellowish, diphtheritic, or necrotic lesions in the oral cavity, which can extend to the proventriculus and gizzard. These painful lesions reduce feed intake and impair overall nutrient absorption.

38. (D) Eimeria tenella
Eimeria tenella is a highly pathogenic coccidian parasite that localizes in the ceca of chickens. It invades the cecal epithelial cells and deeper mucosa, causing severe vascular damage. This leads to the classic clinical presentation of bloody droppings, marked thickening of the cecal wall, and the formation of large hemorrhagic or caseous cecal cores.

39. (B) Eimeria necatrix
Eimeria necatrix is a highly pathogenic species that primarily infects the mid-small intestine of chickens. The parasite causes extensive tissue destruction, characterized grossly by a ballooned intestine with dark red petechiae intermingled with white spots on the serosal surface. This distinctive "salt and pepper" appearance represents focal hemorrhages and clusters of large schizonts.

Click Here to Buy book on Flipkart

40. (C) Liver
Histomoniasis, or blackhead disease, causes severe enterohepatitis in turkeys. The protozoan Histomonas meleagridis migrates from the ceca to the liver, where it produces pathognomonic, depressed, circular, yellowish-green necrotic areas. These "target-like" lesions are highly characteristic of the disease and are accompanied by severe necrotizing typhlitis with caseous cecal cores.

41. (D) Heterakis gallinarum
The transmission of Histomonas meleagridis is facilitated by the cecal nematode Heterakis gallinarum. The fragile protozoon penetrates the ovaries of the female nematode and becomes incorporated into its eggs. This protective environment shields the protozoon, allowing it to survive in the soil for long periods and infect new avian hosts upon ingestion.

42. (A) Intestinal impaction or rupture
Ascaridia galli is a large roundworm that inhabits the small intestine of chickens. Heavy infestations of these nematodes can lead to mechanical blockages in the intestinal lumen. This can cause partial or complete intestinal impaction, mucosal damage, and in severe cases, rupture of the intestinal wall, leading to fatal peritonitis.

43. (A) P-4, Q-1, R-2, S-3
Avian internal parasites have specific anatomical preferences within the host. Capillaria annulata infects the crop and esophagus, while Heterakis gallinarum is found in the ceca. Raillietina cesticillus is a tapeworm that localizes in the small intestine, whereas Syngamus trachea, commonly known as the gapeworm, resides within the tracheal lumen.

44. (B) Ants and beetles
Tapeworms of the genus Raillietina are common intestinal parasites in poultry that require specific intermediate hosts to complete their life cycle. Various species of ants and beetles ingest the tapeworm eggs, allowing the cysticercoids to develop. Chickens become infected by foraging and consuming these arthropod hosts in their environment.

45. (A) Vitamin E
Encephalomalacia, or crazy chick disease, is a neurological condition caused by Vitamin E deficiency in young, rapidly growing chicks. This deficiency leads to lipid peroxidation of membrane lipids, resulting in vascular thrombosis, edema, and hemorrhage within the cerebellum. Affected chicks display ataxia, head tremors, backward falling, and lateral recumbency.

46. (B) Riboflavin (B2)
Riboflavin deficiency in young chicks produces a characteristic clinical presentation known as curled toe paralysis. The lack of this essential vitamin leads to myelin degeneration and swelling of the sciatic nerves. Affected chicks walk on their hocks with their toes curled inward, showing reduced growth and poor feathering.

47. (A) Both A and R are true and R is the correct explanation of A.
Vitamin A deficiency in poultry leads to the formation of small, white, pustule-like lesions in the esophagus and crop mucosa. This occurs because the deficiency causes squamous metaplasia of the mucous gland epithelium, which blocks the gland ducts. This blockage leads to mucus accumulation and the development of retention cysts.

NODIA

48. (C) Selenium
Exudative diathesis is a severe nutritional disease in young chicks characterized by extensive subcutaneous edema, particularly on the breast and abdomen. This condition is caused by a simultaneous deficiency of Vitamin E and selenium. These nutrients act as antioxidants to maintain capillary membrane integrity, and their absence leads to capillary leakage.

49. (B) Nutritional secondary hyperparathyroidism (Rickets)
Formulating poultry diets with an inverted calcium-to-phosphorus ratio, where dietary phosphorus exceeds calcium, triggers nutritional secondary hyperparathyroidism. This mineral imbalance stimulates the parathyroid glands to mobilize calcium from bones. In growing birds, this results in rickets, characterized by soft, rubbery bones and widened, poorly mineralized epiphyseal growth plates.

50. (D) Manganese and choline
Perosis, or slipped tendon, is a nutritional disease in growing poultry characterized by the displacement of the gastrocnemius tendon from the intercondylar groove of the hock joint.

This condition is caused by a deficiency of manganese and choline, which are essential for normal glycosaminoglycan synthesis and matrix development in cartilage.

51. (B) I and III
Rickets in young birds is characterized by poorly mineralized, widened growth plates due to vitamin or mineral imbalances. Similarly, tibial dyschondroplasia involves the persistence of an avascular cartilage plug in the proximal tibiotarsus. In contrast, cage layer fatigue involves the depletion of medullary and cortical bone, rather than excessive bone formation.

52. (C) Marked right ventricular hypertrophy and dilation
Ascites syndrome in fast-growing broilers is driven by pulmonary hypertension, which forces the heart to work harder to pump blood through the lungs. This chronic pressure overload leads to marked right ventricular hypertrophy and subsequent dilation. This right-sided heart failure causes venous congestion and the accumulation of fluid in the abdomen.

53. (A) Turkeys
Round heart disease, or spontaneous cardiomyopathy, is a cardiovascular condition that primarily affects young turkeys, typically between one and four weeks of age. Affected birds show marked enlargement and rounding of the heart, driven by left ventricular dilation and hypertrophy, which leads to sudden death due to acute congestive heart failure.

54. (D) Monosodium urate crystals
Visceral gout is a metabolic disorder characterized by the deposition of insoluble urate crystals on visceral organs, particularly the pericardium, liver capsule, and kidneys. These chalky white deposits consist of needle-like monosodium urate crystals. This condition occurs when renal function is compromised, preventing the normal excretion of uric acid from the body.

55. (B) I and III
Articular gout is directly associated with chronic hyperuricemia and systemic metabolic dysfunction rather than localized trauma in birds with normal blood uric acid levels. Additionally, water deprivation followed by sudden rehydration is a known trigger that precipitates visceral gout, making the statements claiming otherwise scientifically incorrect.

56. (C) Caged, heavy-producing laying hens
Fatty Liver Hemorrhagic Syndrome is a major metabolic disease that causes sudden death in caged, heavy-producing laying hens. High-energy diets combined with limited physical activity lead to excessive fat deposition in the liver. This lipid accumulation makes the liver friable, predisposing it to subcapsular hematomas and fatal intra-abdominal hemorrhage.

57. (D) Biotin
Fatty Liver and Kidney Syndrome is a metabolic disorder that affects young chicks, causing lethargy, hypoglycemia, and pale, fat-laden livers and kidneys. This condition is caused by a nutritional deficiency of biotin, which is an essential cofactor for gluconeogenic enzymes. Supplementing diets with biotin prevents the disease by restoring normal lipid and carbohydrate metabolism.

58. (A) Depletion of structural cortical bone to maintain calcium for eggshells
Cage layer fatigue is a metabolic disease that affects high-producing laying hens kept in cages. The demand for calcium during eggshell formation forces the bird to mobilize calcium from its bones. This chronic mobilization depletes both medullary and structural cortical bone, leading to osteomalacia, skeletal fragility, and paralysis.

NODIA

59. (A) I, II and V
A false layer ovulates normally, but the yolk falls into the peritoneal cavity due to infundibular dysfunction, often leading to egg peritonitis with secondary Escherichia coli infections. In addition, Mycoplasma gallisepticum is a known trigger for salpingitis in laying flocks. However, salpingitis refers to oviduct inflammation, not ovarian inflammation.

60. (B) Early exposure to Infectious Bronchitis Virus
Early exposure of young chicks to nephropathogenic or respiratory strains of Infectious Bronchitis Virus can cause permanent damage to the immature reproductive tract. This infection leads to cystic dilation and non-functional development of the oviduct. As adults, these birds ovulate normally but are unable to produce eggs, becoming clinical false layers.

(C) Escherichia coli
(D) Staphylococcus aureus

2011

194. Ascites syndrome is associated with increased resistance to pulmonary blood flow. Which anatomical feature of the avian lung predisposes it to this resistance?
(A) Highly elastic parabronchial tissue that collapses easily under physiological pressure
(B) Rigid, non-expandable parabronchial network restricting capillary expansion
(C) A functional muscular diaphragm compressing the pulmonary parenchyma during respiration
(D) Excessive, abnormal surfactant production causing extensive capillary alveolar sticking

2008

195. Visceral gout is the deposition of urates on the serosal surfaces. This condition fundamentally reflects:
(A) Hepatic failure
(B) Cardiac failure
(C) Calcium metabolic dysfunction
(D) Renal failure

2017

196. An important pathological consequence of early infectious bursal disease virus (IBDV) infection is the poor response to subsequently administered vaccines. This happens due to:
(A) Destruction of bursa-derived B-lymphocytes
(B) Severe depletion of T-lymphocytes in the thymus
(C) Systematic destruction of circulating heterophils
(D) Complete blockade of host cell viral receptors

2024

197. A layer hen is presented with an enlarged, pendulous abdomen. Post-mortem reveals a cystic left oviduct filled with clear fluid. This anomaly typically occurs due to:
(A) Congenital genetic predisposition unique to heavy breeder strains
(B) Localized, ascending bacterial infection originating from the cloaca
(C) Early neonatal Infectious Bronchitis Virus (IBV) infection arresting normal oviduct development
(D) Prolonged feeding of high calcium diets during rearing phases

2012

198. The pathogenesis of Fowl Cholera in its acute septicemic form involves the massive release of which bacterial component?
(A) Endotoxin
(B) Exotoxin A
(C) Enterotoxin
(D) Botulinum toxin

2007

199. Fowl pox causes proliferative lesions on the non-feathered parts of the skin. The initial stage of these lesions is best classified as:
(A) A fluid-filled vesicle
(B) A raised cellular papule
(C) A superficial skin ulcer
(D) A nodular hyperplasia

2023

200. In evaluating early chick mortality, a firm, severely enlarged, discolored, and unabsorbed yolk sac at 7 days of age strongly indicates:
(A) Normal physiological absorption
(B) Omphalitis
(C) Coccidiosis
(D) Vitamin A deficiency

2010

201. Which specific organ must be preserved in 10% neutral buffered formalin to confirm a diagnosis of Marek's disease histopathologically via the demonstration of lymphomatous infiltrates?
(A) Trachea
(B) Tendon of gastrocnemius
(C) Vagus and sciatic nerves
(D) Crop

2021

202. Necropsy of a turkey reveals severe caseous exudate in the lungs and air sacs. If this is avian chlamydiosis, the causative agent Chlamydia psittaci can be identified cytologically via impression smears using which stain?
(A) Gram staining procedure
(B) Gomori Methenamine Silver stain
(C) Periodic Acid-Schiff stain
(D) Macchiavello or Gimenez stain

2007

203. During an investigation of sudden layer drop and soft-shelled eggs, you suspect Egg Drop Syndrome (EDS-76). The virus causes these defects by primarily replicating in and damaging the:
(A) Shell gland (uterus)
(B) Infundibulum region

Downloaded from www.pdf.tube

pathological signs, certain lesions help differentiate them. Severe subcutaneous edema of the head, neck, and eyelids is a common gross lesion in cases of HPAI. This localized edema is typically less prominent or absent in birds infected with Newcastle disease.

72. (C) Classifying the virus based on its virulence and disease manifestation in the host
Pathotyping is a biological characterization method used to classify avian viruses, such as Newcastle disease virus or avian influenza. This process involves grouping viral isolates based on their virulence, disease severity, and clinical manifestation in susceptible hosts, helping veterinary researchers determine the threat level posed by specific viral strains to poultry.

73. (D) Adenoviridae
The clinical presentation of sudden mortality in young broilers combined with pale, swollen, friable livers containing subcapsular hemorrhages is highly characteristic of inclusion body hepatitis. This disease is caused by fowl adenoviruses, which belong to the family Adenoviridae. The diagnostic finding is the presence of basophilic intranuclear inclusion bodies within necrotic hepatocytes.

74. (A) Ascites
In veterinary pathology, the accumulation of non-inflammatory fluid or transudate within the peritoneal cavity of a bird is termed ascites. This condition often results from increased hydrostatic pressure or decreased oncotic pressure. It is commonly observed in fast-growing broilers as a consequence of pulmonary hypertension and right-sided heart failure.

75. (B) Bollinger bodies
The cutaneous form of fowl pox is characterized by proliferative epithelial lesions on unfeathered skin. Histopathological examination of these skin lesions reveals epidermal hyperplasia and ballooning degeneration. The cells contain large, acidophilic, lipid-rich intracytoplasmic inclusion bodies known as Bollinger bodies, which contain smaller infectious viral particles called Borrel bodies.

76. (B) Fowl pox inclusion bodies are intracytoplasmic, whereas ILT inclusion bodies are intranuclear.
Fowl pox and infectious laryngotracheitis are both viral diseases of poultry that produce distinctive inclusion bodies. Fowl pox, a poxvirus, replicates in the cytoplasm and forms large, acidophilic, intracytoplasmic Bollinger bodies. In contrast, infectious laryngotracheitis virus, a herpesvirus, replicates in the nucleus and produces acidophilic, intranuclear Cowdry type A inclusion bodies.

77. (A) Egg peritonitis caused by Escherichia coli entering through the cloaca
Ascending infections of the avian reproductive tract occur when pathogens migrate upward from the cloaca into the oviduct. A classic example is egg peritonitis, where environmental bacteria, primarily Escherichia coli, ascend through the vagina and uterus. This bacterial migration leads to inflammation of the oviduct and the accumulation of yolk material in the abdominal cavity.

78. (C) It is a viral oncogene carried by Rous sarcoma virus that causes rapid transformation.
The v-src gene is a classic viral oncogene carried by the Rous sarcoma virus, which is an acutely transforming retrovirus. It encodes a constitutively active tyrosine kinase that phosphorylates target host proteins involved in cell signaling pathways. This uncontrolled signaling bypasses normal regulatory mechanisms, leading to rapid neoplastic transformation of infected cells.

79. (D) Turkey coryza (Turkey Rhinotracheitis)
Avian Metapneumovirus is the primary etiological agent responsible for turkey rhinotracheitis, which is commonly referred to as turkey coryza. This highly contagious disease causes acute respiratory signs in turkeys, including sneezing, nasal discharge, rales, and suborbital swelling. It can be complicated by secondary bacterial infections, leading to high flock morbidity.

80. (C) Marek's disease
The asymmetric paralysis of the legs or wings, often described as a dropping wing or the classic dancer's pose, is a pathognomonic clinical sign of Marek's disease. This paralysis results from the neoplastic infiltration of T-lymphocytes into peripheral nerves, such as the sciatic and

NODIA

birds consume indigestible, fibrous materials such as tough, dry grasses, litter, feathers, or foreign objects. These materials physically obstruct the passage of ingesta into the proventriculus, causing fermentation.

120. (B) Central caseous necrosis without calcification, surrounded by giant cells
Avian tuberculosis, caused by Mycobacterium avium, produces chronic, progressive granulomatous lesions in visceral organs like the liver and spleen. Histologically, these granulomas exhibit a characteristic structure consisting of a central core of caseous necrosis that is distinctively non-calcified. This necrotic core is surrounded by an inner layer of epithelioid histiocytes and multinucleated giant cells.

121. (C) Ingestion of contaminated soil or water
Mycobacterium avium subspecies avium is a resilient environmental pathogen capable of surviving for long periods in soil and litter. The primary natural route of transmission in poultry is the fecal-oral pathway, occurring through the ingestion of contaminated feed, soil, water, or feces. Ingested bacilli cross the intestinal mucosa, leading to systemic dissemination and granuloma formation.

122. (D) Narrow, septate, and branching at acute angles
Aspergillus fumigatus is a common opportunistic fungal pathogen causing respiratory infections in birds. Histological examination of infected lung tissue using Gomori methenamine silver staining allows clear visualization of the fungal morphology. The hyphae are characterized by a narrow, uniform diameter, regular septation, and a classic pattern of dichotomous branching at acute angles, approximately forty-five degrees.

123. (A) P-3, Q-2, R-1, S-4
Different toxins exhibit specific tissue tropism and clinical pathology in avian species. Botulinum neurotoxin causes neuromuscular blockade resulting in flaccid paralysis. Aflatoxin B1 targets hepatocytes, causing acute necrosis and chronic biliary hyperplasia. Trichothecene T-2 toxin is highly caustic, producing oral mucosal lesions and severe immunosuppression. Monensin toxicosis impairs intracellular calcium balance, leading to skeletal and cardiac muscle necrosis.

124. (B) Blocking acetylcholine release at the neuromuscular junction
Botulinum toxin, produced by Clostridium botulinum, is a potent neurotoxin responsible for limberneck in birds. Once ingested, the toxin binds specifically to presynaptic membranes of cholinergic nerves. It enzymatically cleaves SNARE proteins, which permanently blocks the calcium-dependent release of the neurotransmitter acetylcholine at the neuromuscular junction, resulting in generalized, progressive flaccid paralysis and respiratory failure.

125. (B) Unidirectional airflow and thin-walled air sacs contacting visceral organs
The unique anatomy of the avian respiratory system utilizes unidirectional airflow and non-vascular air sacs that act as bellows. These thin-walled air sacs extend throughout the body cavity, directly contacting visceral organs and bones. Inhaled respiratory pathogens can easily bypass upper defenses, quickly spread into the coelom, and cross the thin epithelial linings to enter the systemic circulation.

126. (A) Pale, swollen, and mottled with urate-distended tubules
Nephropathogenic strains of infectious bronchitis virus show a strong tropism for the epithelial cells of the renal tubules. Viral replication causes severe interstitial nephritis and tubular degeneration. This damage impairs normal uric acid excretion, causing the kidneys to become grossly enlarged, pale, and mottled, with the tubules and ureters visibly distended with white urate deposits.

127. (A) Both A and R are true and R is the correct explanation of A.
Infectious laryngotracheitis virus, an alphaherpesvirus, is known for its ability to establish lifelong latent infections within the sensory neurons of the trigeminal ganglion. Unattenuated live vaccines can replicate, persist, and undergo latency in vaccinated birds. Under stress, the latent virus can reactivate and shed, potentially initiating outbreaks in susceptible, unvaccinated flock contacts.

128. (D) A severely enlarged, cherry-red liver
Erythroblastosis is a form of avian leukosis characterized by the neoplastic proliferation of immature erythroid cells within the vascular system. Because these erythroblasts accumulate

Click Here to Buy book on Amazon

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

90. (A) Gangrenous dermatitis
Gangrenous dermatitis is a disease of young chickens characterized by severe dermal necrosis and subcutaneous emphysema. The condition typically occurs secondary to immunosuppressive diseases, such as Gumboro or chicken infectious anemia. Opportunistic pathogens, primarily Clostridium or Staphylococcus species, invade the skin, producing necrotizing toxins that cause dark, moist, and crepitant lesions.

91. (B) II only
Vitamin B12 deficiency is not the direct cause of curled toe paralysis in chicks; this specific neurological condition is caused by a deficiency of riboflavin. While other vitamin deficiencies cause severe nervous system signs, associating Vitamin B12 with curled toe paralysis is scientifically incorrect, as B12 deficiency primarily causes anemia and poor hatchability.

92. (B) Riboflavin
Riboflavin deficiency in breeding hen diets has a direct impact on embryonic development. In affected embryos, the feathers fail to rupture their sheaths normally, resulting in a coiled, circular pattern known as "clubbed down." This pathognomonic lesion is often accompanied by dwarfism, edema, and high embryonic mortality during the mid-incubation period.

93. (B) Vitamin A
A high incidence of blood spots within chicken eggs is linked to a dietary deficiency of Vitamin A. This vitamin is essential for maintaining the integrity of mucosal membranes and blood vessels. Its deficiency leads to capillary fragility within the ovarian follicle, causing small hemorrhages during ovulation that deposit blood on the yolk surface.

94. (C) An aviadenovirus (Type II)
Marble spleen disease is an acute, highly fatal respiratory disease of pheasants caused by a Type II aviadenovirus. The virus primarily targets the spleen, causing severe splenomegaly with a characteristic mottled, marble-like appearance due to lymphoid necrosis and hyperplasia. It also induces pulmonary congestion and edema, leading to asphyxiation and sudden mortality.

95. (B) I, III and IV
Mycoplasma gallisepticum is the primary agent of chronic respiratory disease in chickens and is transmitted vertically through the egg. Because Mycoplasma species naturally lack a rigid peptidoglycan cell wall, they are completely resistant to beta-lactam antibiotics like penicillins. Mycoplasma meleagridis, however, primarily affects turkeys and does not spare them.

96. (C) Severe hypoglycemia
Spiking mortality syndrome is a condition of young broiler chickens characterized by sudden, transient peaks in mortality. Just prior to death, affected chicks exhibit profound depression, recumbency, and frequent convulsions. This clinical presentation is caused by severe hypoglycemia, which results from metabolic disturbances that disrupt normal glucose regulation in the liver.

97. (B) Ionophore toxicity (e.g., monensin overdose)
Ingestion of toxic levels of ionophores, such as monensin, causes severe myocardial and skeletal muscle damage in poultry. This toxicity alters intracellular calcium balance, leading to segmental myofiber necrosis. Grossly, this manifests as pale streaking or "tiger-stripe" lesions in skeletal muscles, which corresponds microscopically to classic Zenker's degeneration and necrosis.

98. (D) Lead toxicity
Lead poisoning is a significant toxicological hazard for waterfowl, typically caused by ingesting lead shot or sinkers. Affected birds exhibit severe depression, weakness, and green bile-stained feces. Post-mortem findings are diagnostic, featuring marked erosion and sloughing of the gizzard lining, proventricular impaction, and generalized muscle atrophy due to chronic starvation.

NODIA

99. (A) I, II, III and IV
Reticuloendotheliosis virus carries the v-rel oncogene, while avian leukosis is an exogenous retrovirus requiring integration near host cellular proto-oncogenes. Furthermore, Marek's disease virus replicates to form cell-free infectious particles exclusively in the feather follicle epithelium. However, Marek's disease virus is highly stable and survives for long periods in poultry dust.

the liver. This distinct color change, resulting from extensive biliary stasis and parenchymal necrosis, is not typically seen in Pullorum disease.

138. (D) Non-motile
The vast majority of Salmonella enterica serovars possess peritrichous flagella that confer active motility. However, Salmonella enterica subspecies enterica serovar Gallinarum, including both its Pullorum and Gallinarum biovars, is a major exception. These avian-adapted pathogens are non-motile because they lack functional flagella, a biological characteristic used to differentiate them from motile paratyphoid Salmonella species.

139. (A) Omphalitis
Mushy chick disease is a common hatchery-related condition affecting newly hatched poultry, anatomically referred to as omphalitis. This disease results from bacterial contamination of the unhealed navel, leading to inflammation of the yolk sac. Affected chicks appear weak, sluggish, and exhibit a soft, flabby, distended abdomen with a wet, inflamed, or poorly closed navel.

140. (B) Contaminated eggshells coupled with improper incubator humidity/temperature
Omphalitis develops when opportunistic bacteria, such as Escherichia coli, enter the open navel before complete closure. This condition is strongly predisposed by compromised sanitation in the hatchery or on eggshells, combined with improper incubator temperature and humidity. Suboptimal incubation conditions delay the physiological retraction of the yolk sac and closure of the navel, facilitating bacterial entry.

141. (B) Ubiquitous intracellular proteases (like furin) in most host tissues
The virulence of Newcastle disease virus is determined by the amino acid sequence at the cleavage site of its fusion glycoprotein. Lentogenic strains require extracellular trypsin-like enzymes found only in the respiratory and intestinal tracts. In contrast, highly virulent velogenic strains possess a multi-basic amino acid motif cleaved by ubiquitous intracellular proteases like furin, enabling systemic viral replication.

142. (A) Culturing on blood agar with a Staphylococcus nurse colony
Avibacterium paragallinarum is a fastidious, Gram-negative, rod-shaped bacterium that causes infectious coryza. It requires nicotinamide adenine dinucleotide, also known as V-factor, for growth. Confirmation is achieved by culturing the organism on blood agar alongside a nurse colony of Staphylococcus aureus, which synthesizes and excretes the necessary V-factor, producing satellite colony growth around the nurse streak.

143. (C) Tetanus
Tetanus in birds is a highly rare but distinct neuromuscular disease caused by the neurotoxin tetanospasmin, produced by Clostridium tetani. This spore-forming environmental anaerobe infects deep wounds, releasing the toxin which retrograde-transports to the central nervous system. The toxin blocks inhibitory neurotransmitter release, causing sustained muscular spasms and a characteristic rigid, saw-horse posture.

144. (D) Egg Drop Syndrome (EDS-76)
Egg Drop Syndrome is caused by an atadenovirus that primarily replicates in the epithelial cells of the shell gland, also known as the uterus. The disease is characterized by a sudden, severe drop in egg production in otherwise healthy-looking laying flocks. Affected birds produce thin-shelled, soft-shelled, shell-less, and depigmented eggs due to localized uterine dysfunction.

145. (C) Egg peritonitis
Egg peritonitis is a major cause of mortality in laying hens, characterized by the presence of ectopic yolk material within the peritoneal cavity. This occurs when yolk fails to enter the infundibulum or when the oviduct ruptures. The presence of free yolk induces severe, aseptic or septic, fibrinous inflammation, often complicated by opportunistic bacterial infections.

146. (B) Severe metabolic osteodystrophy causing paralysis in high-yielding caged hens
Cage layer fatigue is a metabolic disease of high-producing laying hens housed in cages, primarily driven by calcium depletion. High demands for eggshell formation combined with inadequate calcium intake force the mobilization of calcium from medullary and cortical bones. This severe bone resorption leads to osteoporosis, fractures of the thoracic vertebrae, and subsequent paralysis.

Downloaded from www.pdf.tube

viscerotropic and velogenic neurotropic strains cause high mortality, the viscerotropic form is uniquely characterized by severe systemic visceral damage. This damage results in prominent, extensive hemorrhagic lesions throughout the gastrointestinal tract, especially in the proventriculus, cecal tonsils, and intestinal lymphoid patches.

111. (C) Liver, spleen, and joints
Systemic amyloidosis in birds develops secondary to chronic antigenic stimulation from persistent bacterial infections, such as tuberculosis or staphylococcosis. This condition is characterized by extracellular deposition of insoluble fibrillar protein aggregates. The primary target organs for this deposition are the liver, spleen, and joint capsules, resulting in progressive organ dysfunction, joint stiffness, and tissue enlargement.

112. (D) Enlarged, pale, firm, and waxy
Amyloid deposition in avian hepatic parenchyma alters the normal structure and physical characteristics of the tissue. Due to extensive extracellular accumulation of amyloid fibrils, the liver becomes grossly enlarged, pale, and remarkably firm. The organ displays a characteristic waxy or translucent appearance on its capsular and cut surfaces, making it highly susceptible to mechanical rupture.

113. (C) Petechiae on the epicardium and abdominal fat
Acute septicemic fowl cholera, caused by Pasteurella multocida, leads to rapid vascular collapse and disseminated intravascular coagulation. The sudden damage to vascular endothelial walls results in widespread petechial and ecchymotic hemorrhages. These pinpoint hemorrhages are most consistently and prominently observed on the epicardial surface of the heart, coronary fat bands, and abdominal fat tissues during necropsy.

114. (B) Enlarged liver with necrotic foci and marginal infarcts
Avian spirochaetosis is an acute septicemic disease of poultry caused by the tick-borne spirochete Borrelia anserina. Systemic infection leads to severe vascular lesions and parenchymal damage. Upon necropsy, the liver appears markedly swollen and congested, containing multifocal areas of necrosis and distinct marginal infarctions resulting from localized thrombosis and ischemic necrosis of the hepatic tissue.

115. (D) Poor litter and abrasive flooring causing mechanical injury
Pododermatitis, commonly known as bumble foot, typically initiates from chronic mechanical irritation or trauma to the plantar epithelium of the footpad. Poor litter management, high moisture levels, and abrasive flooring damage the protective epidermal barrier. This mechanical compromise facilitates the entry of opportunistic environmental pathogens, particularly Staphylococcus aureus, leading to deep ulcerative inflammation and abscessation.

116. (A) Schwann cell proliferation and myelin loss
Curled toe paralysis is a classic nutritional deficiency syndrome in young poultry caused by a lack of riboflavin, also known as vitamin B2. The deficiency leads to progressive degeneration of the peripheral nervous system, particularly the sciatic nerves. Histopathological examination of affected nerves reveals characteristic myelin sheath degeneration, Schwann cell proliferation, and axonal swelling, causing motor dysfunction.

117. (C) Crypt epithelial cells
Eimeria tenella is a highly pathogenic coccidian parasite that selectively infects the cecum of chickens. The parasite invades and replicates within the epithelial cells lining the cecal crypts, rather than the villi. The second-generation schizogony within these crypt cells causes severe cell lysis, leading to mucosal destruction, deep hemorrhage, and classic bloody cecal cores.

NODIA

118. (A) Sporozoite
The lifecycle of Eimeria species begins when host chickens ingest sporulated oocysts from the environment. Inside the digestive tract, mechanical and enzymatic processes trigger excystation, releasing active sporozoites. These mobile, infectious forms possess specialized apical complexes designed to recognize, attach to, and actively penetrate the plasma membrane of intestinal epithelial cells to initiate intracellular replication.

119. (D) Ingestion of tough grasses or foreign bodies
Crop impaction, also known as sour crop, is a form of upper gastrointestinal tract stasis characterized by the accumulation of food and fluid. This condition commonly occurs when

brachial plexuses, causing nerve swelling and loss of function.

81. (B) Central chromatolysis of neurons and non-purulent encephalomyelitis
Avian encephalomyelitis is a viral infection that targets the central nervous system of young chicks. Histopathological examination of brain and spinal cord tissues reveals non-purulent encephalomyelitis, characterized by perivascular cuffing and glial nodules. The diagnostic finding is central chromatolysis of neurons, particularly in the nuclei of the medulla oblongata.

82. (D) Severe acute catarrhal inflammation of the upper respiratory tract with facial edema
Infectious coryza is an acute respiratory disease of chickens caused by the bacterium Avibacterium paragallinarum. The hallmark clinical and pathological presentation is severe, acute, catarrhal inflammation of the nasal passages and infraorbital sinuses, accompanied by conspicuous facial edema and a foul-smelling nasal discharge, primarily affecting the upper respiratory tract.

83. (A) P-2, Q-4, R-1, S-3
Pathogenic agents in poultry have specific tissue tropisms. Eimeria acervulina targets the duodenal loop of the small intestine, while Mycoplasma synoviae infects joints and tendon sheaths. Infectious laryngotracheitis virus replicates in the tracheal epithelium, and Histomonas meleagridis causes necrotizing lesions in both the ceca and liver of susceptible birds.

84. (C) M. synoviae produces creamy to caseous yellow joint exudate, while viral arthritis exhibits clear fluid or fibrosis.
Infectious synovitis caused by Mycoplasma synoviae can be differentiated from viral arthritis based on the nature of the joint fluid. Mycoplasma synoviae infections produce a creamy, viscid, or caseous yellowish-gray exudate within the joints and tendon sheaths. In contrast, viral arthritis, caused by reovirus, is characterized by clear or serosanguinous fluid and fibrosis.

85. (A) Histomoniasis
The passage of bright yellow, sulfur-colored feces is a clinical sign of histomoniasis in turkeys. This distinctive coloration is caused by severe hepatic dysfunction and parenchymal necrosis as the protozoan Histomonas meleagridis destroys liver tissue. The damaged liver is unable to process bile pigments normally, leading to their excretion in the droppings.

86. (D) Hypoxia induces pulmonary arterial vasoconstriction, leading to right ventricular hypertrophy and subsequent heart failure.
At high altitudes, the reduced partial pressure of oxygen causes chronic hypoxia in fast-growing broilers. This hypoxia triggers pulmonary arterial vasoconstriction, increasing resistance to blood flow. To overcome this resistance, the heart undergoes right ventricular hypertrophy, which eventually progresses to right-sided heart failure, venous congestion, and the accumulation of ascites fluid.

87. (A) Both A and R are true and R is the correct explanation of A.
Intestinal coccidiosis is a well-established predisposing factor for necrotic enteritis in chickens. The damage to the intestinal mucosa caused by Eimeria replication, combined with plasma leakage into the lumen, provides a nutrient-rich environment. This microenvironment promotes the rapid proliferation of Clostridium perfringens, which subsequently produces the toxins that cause necrotic enteritis.

88. (C) Alpha toxin or NetB toxin
Necrotic enteritis in poultry is primarily mediated by the action of potent extracellular toxins produced by Clostridium perfringens. Alpha toxin, a phospholipase C, and NetB toxin, a pore-forming toxin, are critical virulence factors. These toxins cause rapid enzymatic degradation of the intestinal mucosal epithelium, leading to confluent mucosal necrosis and pseudomembrane formation.

89. (B) Eimeria acervulina
Eimeria acervulina is correct because it infects the upper small intestine, especially the duodenum, producing characteristic transverse white plaques that give a "ladder-like" appearance at necropsy. These lesions result from heavy parasite development within the intestinal epithelium. Eimeria tenella primarily affects the caeca, Eimeria necatrix causes severe lesions in the mid-intestine, and Eimeria maxima mainly produces thickened, dilated intestines with orange mucoid contents.

Click Here to Buy book on Flipkart

in massive numbers inside the sinusoids of visceral organs, affected birds develop pronounced hepatosplenomegaly. The liver becomes remarkably enlarged, soft, and displays a characteristic uniform, bright cherry-red color due to vascular engorgement.

129. (C) Diaphyseal thickening of long bones due to extreme periosteal bone formation
Avian osteopetrosis is a neoplastic-like disease of the skeletal system induced by certain retroviruses of the avian leukosis group. The pathogenesis involves abnormal osteoblast activity, resulting in excessive, accelerated bone deposition. This occurs primarily along the diaphysis of the long bones, leading to progressive, symmetrical thickening of the shanks and limbs, giving a characteristic thick-legged appearance.

130. (B) Vasogenic brain edema and inflammation affecting the central nervous system
The classic transient paralysis syndrome seen in Marek's disease is an acute neurological manifestation of the infection. Unlike the chronic paralytic form caused by direct lymphocytic infiltration of peripheral nerves, this temporary state is primarily mediated by immune-mediated vascular injury within the central nervous system, leading to acute vasogenic brain edema and inflammatory vasculitis.

131. (D) Keratoconjunctivitis
High atmospheric ammonia levels in poorly ventilated poultry facilities directly dissolve in the moisture of the eyes, forming ammonium hydroxide. This alkaline chemical is highly corrosive to mucosal membranes. It causes localized tissue irritation, leading to epithelial damage, conjunctivitis, blepharospasm, and corneal ulceration, clinically recognized as keratoconjunctivitis, which serves as a key indicator of poor ventilation.

132. (A) Subcutaneous edema around the periorbital tissues
Avian metapneumovirus is the primary causative agent of swollen head syndrome in chickens and turkeys. The virus causes acute upper respiratory tract inflammation. This inflammatory response leads to significant accumulation of subcutaneous inflammatory exudate and fluid in the periorbital and facial tissues, producing a characteristic symmetrical swelling or spectacle-like appearance around both eyes.

133. (C) Scaly leg disease
Cnemidocoptes mutans is a microscopic, burrowing mite that infects the unfeathered skin of birds, primarily on the legs and feet. The mites tunnel deep within the epidermis of the metatarsus, causing mechanical irritation, hyperkeratosis, and inflammatory exudates. This process results in thickened, crusty, raised scales, a condition clinically referred to as scaly leg disease.

134. (A) IgY
Immunoglobulin Y, the avian functional equivalent of mammalian immunoglobulin G, is the primary antibody class responsible for systemic humoral immunity in birds. During egg formation, immunoglobulin Y is selectively transferred from the maternal serum across the follicular epithelium into the yolk. This transfer provides essential systemic passive immunity to the developing chick during early life.

135. (D) Newcastle Disease
The junction between the proventriculus and the gizzard is highly sensitive to pathological damage during systemic viral infections. Highly virulent, velogenic strains of Newcastle disease virus exhibit strong vascular tropism and cause endothelial injury. This leads to characteristic necrotic, diphtheritic, or band-like hemorrhagic lesions concentrated specifically at this mucosal transition zone, serving as a classic diagnostic indicator.

136. (B) Dark, dry breast muscle and prominent ureteral urates
Dehydration in poultry causes a rapid loss of intracellular and extracellular fluids, leading to hemoconcentration. Upon post-mortem examination, this state is characterized by dark, dry, and sticky skeletal muscles, particularly visible in the breast region. Furthermore, impaired renal clearance leads to the accumulation and visible deposition of thick white urates within the kidneys and ureters.

137. (C) Fowl Typhoid typically produces a distinct, swollen, bronze-green discolored liver.
Although both diseases are caused by Salmonella enterica serovars, Fowl Typhoid, caused by Salmonella Gallinarum, presents differently in adult birds. Acute septicemic Fowl Typhoid causes severe hepatomegaly accompanied by a characteristic coppery or bronze-green sheen on

Downloaded from www.pdf.tube

100. (C) Thymus and bursa of Fabricius
Reticuloendotheliosis virus infection in chickens can manifest as a chronic runting-stunting syndrome. Pathologically, affected birds exhibit severe weight loss, abnormal feathering, and profound immunologic compromise. This runting syndrome is characterized by severe atrophy of the primary lymphoid organs, specifically the thymus and the bursa of Fabricius, due to viral-mediated lymphoid depletion.

101. (A) "Blow-out" or vent prolapse
Vent prolapse, commonly referred to as a "blow-out" in commercial laying flocks, is a condition where the tissues of the oviduct and cloaca remain everted through the vent after oviposition. This exposed, hyperemic tissue attracts attention from other birds, frequently triggering severe cannibalism and high mortality due to pick-outs.

102. (D) Uterus (shell gland) or vagina
An egg-bound condition occurs when a fully or partially formed egg becomes physically lodged within the avian reproductive tract. This pathology is most commonly localized in the distal segments of the oviduct, specifically the uterus or the vagina. It can result from hypocalcemia, pelvic trauma, or the production of abnormally large eggs.

103. (A) II, V, IV, I, III
During egg formation, the ovum released from the ovary traverses the specialized segments of the single left oviduct in a precise chronological sequence. It first enters the infundibulum, followed by the magnum, the isthmus, the uterus, and finally the vagina, where each anatomical region contributes specific structural layers to the complete egg.

104. (B) The chalazae and albumen
The magnum is the longest and most glandular segment of the avian oviduct. As the ovum passes through this region, the glandular epithelial cells secrete bulk proteins that form the chalazae and the thick and thin layers of the albumen. This protein deposition provides essential nutrients and protective cushioning for the developing embryo.

105. (D) Chicken Astrovirus (CAstV)
White chick syndrome is a recently recognized pathology associated with vertical transmission of chicken astrovirus. Affected breeder flocks experience a transient drop in hatchability, while offspring show weakness, pale plumage, and green-colored livers. The virus causes severe degeneration of the intestinal epithelium and lymphoid organs, leading to significant economic losses.

106. (D) Avian hepatitis E virus
Big Liver and Spleen Disease is a major infectious disease affecting broiler breeders and laying hens, characterized by splenomegaly, hepatomegaly, and decreased egg production. The causative agent is avian hepatitis E virus, a non-enveloped, single-stranded, positive-sense RNA virus. This virus primarily targets hepatocytes and splenic macrophages, causing characteristic organ enlargement and vascular congestion.

107. (A) III, I, II, IV, V
Standard viral isolation for Newcastle disease virus begins with sterile collection of tissues like trachea, brain, and spleen on ice. These tissues undergo homogenization to create a ten percent suspension. This suspension is inoculated into the allantoic cavity of embryonated eggs. Following embryo death or incubation, allantoic fluid is harvested and tested via spot hemagglutination.

108. (C) More than 90 hours
The virulence of Newcastle disease virus strains is categorized using the mean death time of minimum lethal doses in embryonated chicken eggs. Lentogenic strains are low-virulence pathotypes that cause mild or subclinical respiratory infections, requiring more than ninety hours to kill embryos. This slow mortality rate distinguishes them from mesogenic and highly virulent velogenic strains.

NODIA

109. (B) Bordetella avium
Infection of turkeys with Bordetella avium leads to bordetellosis, commonly referred to as turkey coryza. This upper respiratory tract disease is characterized by the colonization of ciliated epithelium, leading to severe tracheal damage, loss of ciliary function, and mucosal remodeling. This pathology results in a thickened, rigid, and narrowed tracheal lumen filled with thick caseous exudate.

110. (A) Hemorrhagic gastrointestinal lesions
Newcastle disease virus exhibits different pathotypes with distinct clinical and pathological presentations. While both velogenic

147. (D) Infectious Laryngotracheitis
Infectious laryngotracheitis is an acute, highly contagious respiratory disease of chickens caused by a gallid alphaherpesvirus. The virus replicates in and destroys the epithelial cells lining the trachea and larynx. This severe mucosal destruction leads to hemorrhage, diphtheritic membrane formation, and casts, causing affected birds to display dyspnea, gasping, and expectoration of bloody mucus.

148. (A) MD peaks around 12-24 weeks; ALV rarely occurs before 14-16 weeks.
Age of onset is a valuable clinical tool for distinguishing neoplastic diseases in chickens. Marek's disease, caused by a herpesvirus, typically manifests in young birds, with clinical signs and mortality peaking between twelve and twenty-four weeks of age. In contrast, lymphoid leukosis, caused by a retrovirus, has a longer incubation period and rarely occurs before sexual maturity.

149. (C) Aflatoxin
Chronic ingestion of aflatoxins, produced by Aspergillus species, causes severe hepatotoxicity in poultry. The toxin undergoes bioactivation in hepatocytes, leading to cellular injury, lipid accumulation, and necrosis. Over time, this persistent toxicity triggers a regenerative and inflammatory response, characterized histologically by marked proliferation of the bile duct epithelium and progressive fibrosis in the periportal regions.

150. (A) Loops of Henle
The avian renal system is anatomically unique, containing a mixture of reptilian-type and mammalian-type nephrons. The majority of these nephrons lack loops of Henle, which limits the kidney's ability to concentrate urine. Consequently, birds rely heavily on active tubular secretion of uric acid. Any compromise to renal function rapidly leads to uric acid accumulation and visceral gout.

151. (D) Pyruvate carboxylase
Fatty Liver and Kidney Syndrome in young chickens is a metabolic disorder caused by a dietary deficiency of biotin. Biotin is a crucial cofactor for pyruvate carboxylase, a key rate-limiting enzyme in gluconeogenesis. A deficiency in this enzyme impairs the synthesis of glucose from non-carbohydrate precursors, leading to severe hypoglycemia, altered lipid metabolism, and lipid accumulation.

152. (B) Massive hypertrophy of the bursa of Fabricius
Malabsorption syndrome, also known as infectious stunting syndrome, is a multifactorial disease of young broiler chickens characterized by severe growth depression and poor feathering. Classic gross lesions include pale, distended intestines containing undigested feed, pancreatic atrophy, and skeletal abnormalities. However, massive hypertrophy of the bursa of Fabricius is not associated with this syndrome, as bursal atrophy is typically observed.

153. (C) Lymphoid leukosis
Lymphoid leukosis is a neoplastic disease of semi-mature and mature chickens caused by avian leukosis retroviruses. The transformation of target cells occurs exclusively within the follicles of the bursa of Fabricius. Metastasis leads to nodular tumor masses in visceral organs, composed of a highly uniform population of large lymphoblasts, which histologically distinguishes this disease from Marek's disease.

154. (D) Avian spirochaetosis
Avian spirochaetosis is a severe, septicemic disease of poultry caused by the spirochete Borrelia anserina. Because the primary vector for transmitting this pathogen is the fowl tick, Argas persicus, the disease is commonly referred to as fowl tick fever. Tick transmission occurs during feeding, introducing the bacteria directly into the bloodstream, where it multiplies rapidly.

155. (A) Permanent immunosuppression from destruction of precursor B-cells
The bursa of Fabricius is the primary site of B-lymphocyte development in young birds. Infectious bursal disease virus targets and destroys actively dividing IgM-positive B-cells within the bursal follicles. When infection occurs early in life, prior to two weeks of age, it results in permanent destruction of these precursor B-cells, leading to lifelong systemic immunosuppression.

156. (B) Pectoral and thigh muscle hemorrhages
Infectious bursal disease, also known as Gumboro disease, exhibits clinical and pathological similarities to other septicemic diseases of poultry. However, acute cases are uniquely characterized by significant vascular damage in skeletal muscles. During necropsy,

Click Here to Buy book on Amazon

affected birds consistently display prominent, widespread petechial or ecchymotic hemorrhages in the pectoral and thigh muscles, which aids in distinguishing it from Newcastle disease.

157. (B) Infectious laryngotracheitis virus
Infectious laryngotracheitis virus is a highly contagious alphaherpesvirus that infects the upper respiratory tract of chickens. Following the acute clinical phase of infection, the virus travels via sensory nerve fibers to the trigeminal ganglion. It establishes a lifelong latent infection within these neurons, from which it can periodically reactivate and shed under stress conditions.

158. (A) Histomoniasis
Histomoniasis, commonly known as blackhead disease, is caused by the protozoan parasite Histomonas meleagridis. The parasite replicates in the ceca, causing severe bilateral necrotic typhlitis and the formation of cheesy, laminated cecal cores. It then migrates via the portal circulation to the liver, producing characteristic circular, depressed, target-like necrotic lesions, which are pathognomonic for this disease.

159. (C) Biotin deficiency
Torticollis, or wry neck, is a clinical sign characterized by abnormal twisting of the neck, typically resulting from neurological or vestibular dysfunction. This condition can be caused by viral infections like Newcastle disease, vitamin E deficiency leading to encephalomalacia, or localized bacterial infections in the cranial cavity. However, biotin deficiency causes dermatitis and perosis, not torticollis.

160. (D) Marek's Disease
Marek's disease is a lymphoproliferative disease of chickens caused by a cell-associated herpesvirus. Neoplastic T-lymphocytes infiltrate various peripheral nerves, leading to loss of striations and marked unilateral or bilateral nerve thickening, particularly of the sciatic and vagus nerves. In addition, these neoplastic cells form solid lymphomatous tumor masses in visceral organs, including the ovary, liver, and lungs.

161. (C) A pleomorphic neoplastic infiltrate of lymphocytes, plasma cells, and macrophages
Marek's disease and lymphoid leukosis both produce visceral tumors, but they differ significantly under histopathological examination. Marek's disease lesions are characterized by a pleomorphic cellular infiltrate, consisting of a mixture of T-lymphoblasts, small, medium, and large lymphocytes, plasma cells, and macrophages. In contrast, lymphoid leukosis lesions are highly monomorphic, consisting entirely of uniform lymphoblasts.

162. (B) Mycoplasma gallisepticum
Mycoplasma gallisepticum is a primary pathogen of the avian respiratory system. The bacteria possess a specialized attachment organelle that binds specifically to receptors on the ciliated tracheal epithelial cells. This attachment causes progressive loss of cilia and epithelial desquamation. This mucosal damage impairs the mucociliary escalator, predisposing the bird to secondary bacterial infections like Escherichia coli.

163. (A) P-2, Q-4, R-1, S-3
Avian metabolic and nutritional disorders are linked to specific physiological imbalances. Perosis is caused by manganese or choline deficiencies, resulting in tendon slippage. Ascites syndrome is triggered by hypoxia and rapid growth, leading to pulmonary hypertension. Exudative diathesis results from a deficiency in vitamin E or selenium. Visceral gout is caused by high calcium-protein diets and water deprivation.

NODIA

164. (A) Fowl adenovirus serotype 4
Hydropericardium syndrome, also known as Angara disease, is an infectious disease primarily affecting young broiler chickens. It is characterized by high mortality and the accumulation of a clear or straw-colored fluid within the pericardial sac. The principal causative agent is fowl adenovirus serotype four, which targets hepatocytes and endothelial cells, causing acute hepatitis and vascular leakage.

165. (C) Wet pox
The diphtheritic or wet form of fowl pox is caused by an avipoxvirus infecting the upper digestive and respiratory tracts. The virus replicates in epithelial cells, leading to hyperplasia and ballooning degeneration. This results in the formation of elevated, yellowish, diphtheritic plaques on the mucous membranes. These plaques are firmly attached and leave bleeding ulcers when removed.

166. (A) Vitamin A lesions are expressible pustules, while pox lesions are firmly attached fibrinous plaques.
Although both conditions produce lesions in the oral cavity and esophagus, their physical nature differs. Vitamin A deficiency causes squamous metaplasia of mucous glands, resulting in small, elevated, white nodules containing keratinized debris that can be easily squeezed out as pustules. Conversely, wet pox lesions are firmly attached, diphtheritic, fibrinous membranes that leave a bleeding surface when scraped.

167. (C) A is true but R is false.
Hemorrhagic enteritis of turkeys is caused by a turkey adenovirus. While the disease indeed causes sudden death with bloody feces, the virus does not directly replicate in or destroy the intestinal epithelial cells. Instead, the virus replicates primarily in the spleen, and the intestinal pathology is mediated by an acute, systemic immune-mediated vascular reaction that triggers massive hemorrhage.

168. (B) Infectious Bronchitis Virus
Infectious bronchitis virus is a coronavirus with a strong tropism for both the respiratory tract and the urogenital system of hens. When it infects the oviduct, it damages the epithelial lining of the magnum and uterus. This damage leads to a loss of internal egg quality and the production of misshapen, thin, rough, or chalky-shelled eggs.

169. (C) Hepatocellular triglyceride accumulation leading to capsular rupture and hemorrhage
Fatty Liver Hemorrhagic Syndrome is a metabolic disorder affecting high-producing laying hens, driven by excessive energy intake. The liver undergoes severe lipidosis, characterized by massive accumulation of triglycerides within hepatocytes. This lipid overload structurally weakens the parenchymal architecture and the hepatic capsule, predisposing the organ to spontaneous rupture, subcapsular hematoma, and fatal abdominal hemorrhage.

170. (D) Chlamydiosis
Avian chlamydiosis is caused by Chlamydia psittaci, a highly infectious intracellular bacterium. Infected birds shed large quantities of the organism in respiratory secretions and feces. During necropsy, inhalation of aerosolized dust or contact with fluids from lesions like fibrinous airsacculitis can transmit the pathogen to humans, causing psittacosis, a severe and potentially life-threatening zoonotic disease.

171. (A) Creamy, viscous exudate in the keel bursa and joint spaces
Mycoplasma synoviae is a significant pathogen of poultry that causes systemic infection with a strong tropism for synovial membranes. During the acute phase of infection, it induces severe inflammatory changes within the joints and tendon sheaths. This leads to the accumulation of a characteristic creamy, viscous, yellow-to-gray inflammatory exudate within the keel bursa and hock joint spaces.

172. (B) Neutrophils
In avian hematology, heterophils are the most abundant granulocytic white blood cells in the peripheral circulation of most healthy birds. They are the primary cellular responders during acute inflammatory processes and bacterial infections. Heterophils function through phagocytosis and degranulation, serving as the direct functional and physiological equivalent of mammalian neutrophils in the host defense system.

173. (B) Avian heterophils lack significant myeloperoxidase, relying on non-oxidative mechanisms for killing.
Although avian heterophils are the functional equivalents of mammalian neutrophils, they possess distinct biochemical differences. Most notably, heterophils lack significant levels of the enzyme myeloperoxidase, which prevents them from producing the oxidative burst characteristic of mammalian neutrophils. Instead, they rely primarily on non-oxidative, oxygen-independent bactericidal mechanisms, such as cationic proteins, lysozymes, and acid hydrolases.

174. (A) Ischemic necrosis of the supracoracoideus muscle due to compression
Deep pectoral myopathy, also known as green muscle disease, is an ischemic necrosis of the supracoracoideus muscle in heavy, fast-growing poultry. Vigorous wing flapping causes this muscle to expand, but its rigid fascial sheath and the overlying sternum restrict swelling. This restriction increases tissue pressure, which occludes the local blood supply, causing ischemia and necrosis.

Downloaded from www.pdf.tube

2024

41. The pathogenesis of optic nerve compression in growing animals with Vitamin A deficiency is due to:
(A) Primary inflammatory demyelination of the optic nerve
(B) Complete bilateral retinal detachment
(C) Direct toxic chemical neuritis
(D) Impaired sphenoid bone remodeling

2014

42. Compared to normal respiratory epithelium, the metaplastic epithelium seen in Vitamin A deficiency is:
(A) Keratinized and lacks cilia
(B) Non-keratinized and ciliated
(C) Highly glandular
(D) Pseudostratified and mucin-rich

2021

43. Which of the following conditions is least relevant to Vitamin A deficiency?
(A) Xerophthalmia
(B) Osteomalacia
(C) Nyctalopia
(D) Squamous metaplasia

2006

44. Identify the INCORRECT pair regarding Vitamin A forms and their physiological functions.
(A) Retinal : Biochemical mediator of vision
(B) Beta-carotene : Active visual pigment
(C) Retinoic acid : Epithelial differentiation
(D) Retinol : Systemic transport and storage form

2002

45. Match the Vitamin A deficiency lesion with the affected tissue or organ:
List I: (Lesion)
P. Squamous metaplasia
Q. Nyctalopia
R. Optic nerve constriction
S. Microphthalmia
List II: (Affected Tissue/Organ)
1. Retina
2. Sphenoid bone foramen
3. Fetal ocular development
4. Respiratory and glandular ducts
(A) P-4, Q-1, R-2, S-3
(B) P-1, Q-4, R-2, S-3
(C) P-4, Q-1, R-3, S-2
(D) P-3, Q-1, R-2, S-4

NODIA

2010

46. Vitamin D deficiency in growing animals results in defective bone mineralization known as:
(A) Osteomalacia
(B) Osteoporosis
(C) Rickets
(D) Osteopetrosis

2006

47. The fundamental pathological mechanism underlying both rickets and osteomalacia is:
(A) Excessive osteoclastic bone resorption
(B) Failure of collagen matrix synthesis
(C) Neoplastic transformation of osteoblasts
(D) Defective mineralization of osteoid

2021

48. Consider the following statements regarding Vitamin D and bone pathology:
I. Vitamin D promotes intestinal absorption of calcium and phosphorus.
II. Rickets affects endochondral ossification at the physis.
III. Osteomalacia involves unmineralized osteoid accumulating on adult trabeculae.
IV. The active form of Vitamin D is 25-hydroxycholecalciferol.
V. Rickets is characterized by widened, irregular epiphyseal plates.
Which of the above statements are correct?
(A) II, III, IV, and V
(B) I, II, and IV
(C) I, II, III, and V
(D) I, III, IV, and V

2022

49. The characteristic "rachitic rosary" lesion observed in rickets is caused by:
(A) Severe hemorrhagic lesions at the costochondral junctions
(B) Enlargement of costochondral junctions due to accumulation of unmineralized osteoid and cartilage
(C) Multiple healing fracture calluses along the rib shafts
(D) Extensive metastatic calcification of the intercostal muscles

2023

50. Microscopically, the epiphyseal plate in an animal with rickets demonstrates:
(A) Premature physeal closure with complete mineralization
(B) Massive ischemic necrosis within the resting cartilage
(C) Progressive depletion of all functional

maturation and differentiation of antibody-producing B-lymphocytes. The organ grows rapidly after hatching, reaching its maximum size relative to the chick's body weight between three and eight weeks of age. Following this peak, it undergoes physiological involution as the bird approaches sexual maturity.

185. (C) Severe hemorrhagic tracheitis with blood clots
Mycoplasma gallisepticum is the primary agent of chronic respiratory disease in chickens, causing catarrhal inflammation of the sinuses, trachea, and air sacs. However, severe hemorrhagic tracheitis containing blood clots is not typical. This severe, bloody presentation is instead a classic and pathognomonic lesion of infectious laryngotracheitis, helping to clinically distinguish these two respiratory diseases during post-mortem examination.

186. (D) T-2 toxin
T-2 toxin is a highly toxic trichothecene mycotoxin produced by various Fusarium species. The toxin possesses a strongly caustic nature, causing direct contact necrosis of epithelial surfaces. Ingestion leads to the development of painful, necrotic lesions in the oral cavity, proventriculus, and gizzard, combined with severe lymphoid depletion in immune organs, resulting in profound systemic immunosuppression.

187. (A) Aspergillus fumigatus
Brooder pneumonia is a common, acute respiratory disease of young chicks and poults caused by the inhalation of large numbers of Aspergillus fumigatus spores. This fungal contamination typically originates from damp, warm hatchery environments or moldy litter. The infection leads to the development of widespread, yellow-white granulomatous nodules within the lungs and air sacs, causing high mortality.

188. (B) Wing droop and leg paresis
Lead poisoning in waterfowl typically occurs following the ingestion of spent lead shot from wetlands. Lead is absorbed into the bloodstream, where it interferes with hemoglobin synthesis and causes neuromuscular dysfunction. Affected birds consistently display progressive peripheral neuropathy, characterized by characteristic bilateral wing droop, leg weakness or paresis, and an inability to stand or fly.

189. (B) A hierarchy of large, yolk-filled follicles
The avian ovary is highly active and functionally dynamic in laying hens. Under normal physiological conditions, a mature, active ovary contains a distinct hierarchy of developing follicles. These follicles range in size from small, white ova to several large, yellow, yolk-filled preovulatory follicles. This highly organized arrangement is absent in non-laying hens or those with reproductive dysfunction.

190. (A) It acts as a paratenic host, harboring infected Heterakis eggs in its tissues.
Histomonas meleagridis is a fragile protozoan parasite that cannot survive long in the environment on its own. To persist and spread, it relies on the egg of the cecal nematode Heterakis gallinarum. Earthworms ingest these nematode eggs containing the protozoan, acting as a paratenic host by harboring the viable larvae in their tissues until consumed by birds.

191. (C) Multiple species of Eimeria as immunity is species-specific
Coccidiosis control in poultry utilizing live vaccines requires the inclusion of multiple Eimeria species. This requirement exists because host immunity to coccidia is strictly species-specific, meaning that exposure to one species, such as Eimeria tenella, provides no protective cross-immunity against others like Eimeria acervulina. Therefore, vaccines must contain all relevant species to ensure complete protection.

192. (D) Eimeria acervulina
Different Eimeria species infect specific regions of the avian intestinal tract. Eimeria acervulina is a moderately pathogenic species that selectively colonizes the duodenal loop and upper jejunum. The parasite replicates in the epithelial cells of the villi, producing characteristic, transversely oriented, white-to-gray plaques or bands that are highly visible on the mucosal surface during necropsy.

193. (C) Escherichia coli
Egg peritonitis is a major cause of mortality in commercial laying hens, characterized by the presence of yolk material in the peritoneal cavity. While this yolk initially causes sterile inflammation, it quickly becomes infected by opportunistic bacteria. Escherichia coli is the most frequently isolated pathogen, causing a

Click Here to Buy book on Flipkart

severe, acute or chronic, fibrinous peritonitis and systemic septicemia.

194. (B) Rigid, non-expandable parabronchial network restricting capillary expansion
Ascites syndrome, or pulmonary hypertension syndrome, is a major metabolic disease of fast-growing broilers. The avian lung is a highly rigid, non-expandable structure, and its capillaries are embedded within a dense, inextensible tissue network. Consequently, these pulmonary blood vessels cannot expand to accommodate the increased cardiac output required during rapid growth, leading to pulmonary hypertension and right-sided heart failure.

195. (D) Renal failure
Visceral gout is characterized by the abnormal deposition of sodium urate crystals on the serosal surfaces of internal organs, particularly the pericardium, liver, and kidneys. Because birds excrete nitrogenous waste primarily as uric acid via active renal tubular secretion, any factor causing severe renal dysfunction or failure impairs this excretory pathway, leading to hyperuricemia and systemic crystal deposition.

196. (A) Destruction of bursa-derived B-lymphocytes
Infectious bursal disease virus exhibits a strong tropism for immature, IgM-bearing B-lymphocytes within the bursa of Fabricius. When infection occurs early in life, the virus replicates extensively in these cells, causing severe lytic necrosis. This destruction prevents the normal development and maturation of the humoral immune system, resulting in a poor response to subsequently administered flock vaccines.

197. (C) Early neonatal Infectious Bronchitis Virus (IBV) infection arresting normal oviduct development
A cystic left oviduct, or water belly, is a common congenital or acquired abnormality in laying hens. This condition is frequently a long-term consequence of early exposure to certain nephropathogenic or uropathogenic strains of infectious bronchitis virus during the first week of life. The virus damages the immature oviduct, preventing normal development and resulting in fluid accumulation.

NODIA

198. (A) Endotoxin
Acute septicemic fowl cholera is caused by Pasteurella multocida, a Gram-negative bacterium. The pathogenesis of this rapid and fatal disease is driven by the massive multiplication of bacteria in the bloodstream. Upon bacterial lysis, large quantities of lipopolysaccharide endotoxin are released into circulation, triggering a cascade of vascular damage, disseminated intravascular coagulation, shock, and sudden death.

199. (D) A nodular hyperplasia
Fowl pox virus exhibits a strong affinity for epithelial cells of the epidermis. Following introduction through skin abrasions or mechanical vectors, the virus replicates within these cells, inducing hyperplasia and swelling. Consequently, the earliest visible stage of the cutaneous lesion is characterized by a firm, raised, localized nodular hyperplasia, rather than a fluid-filled vesicle.

200. (B) Omphalitis
Under normal physiological conditions, a chick's yolk sac is almost completely absorbed by seven days of age. The presence of a firm, enlarged, discolored, and unabsorbed yolk sac at this stage indicates yolk sac infection, also known as omphalitis. This bacterial contamination prevents normal nutrient absorption and induces severe, localized inflammatory changes, leading to early chick mortality.

201. (C) Vagus and sciatic nerves
Marek's disease is a lymphoproliferative disease characterized by the neoplastic infiltration of T-lymphocytes into peripheral nerves and visceral organs. To confirm this diagnosis histopathologically, samples of the major peripheral nerves, specifically the vagus and sciatic nerves, must be preserved. Histological examination of these tissues reveals characteristic lymphocytic infiltration, demyelination, and Schwann cell proliferation.

202. (D) Macchiavello or Gimenez stain
Chlamydia psittaci is an obligate intracellular bacterium that does not stain well with standard Gram staining. To visualize the characteristic elementary and reticulate bodies within the cytoplasm of infected cells, specialized staining methods must be used. The Gimenez or Macchiavello stains are highly effective, staining the infectious elementary bodies a bright red against a contrasting blue background.

203. (A) Shell gland (uterus)
Egg Drop Syndrome is caused by an adenovirus that displays a strong tissue tropism for the reproductive tract of laying hens. The virus primarily replicates within the epithelial cells of the shell gland, also known as the uterus. This localized replication causes severe cell lysis and inflammatory changes, directly impairing calcium deposition during shell formation.

204. (B) Highly variable and not always present, despite the disease name
Histomoniasis is commonly known as blackhead disease because affected birds, especially turkeys, sometimes develop a dark, cyanotic head due to poor oxygenation and circulatory collapse. However, this clinical sign is highly variable and often absent in many cases. Diagnostic confirmation relies instead on identifying the characteristic necrotizing lesions in the ceca and liver.

205. (B) Thymus
The avian immune system consists of two primary lymphoid organs responsible for lymphocyte development. While the bursa of Fabricius is the site for B-cell maturation, the thymus is the primary organ responsible for the development, differentiation, and selection of T-lymphocytes. It consists of multiple lobes located along the neck of the bird, adjacent to the jugular vein.

206. (A) H5 and H7
Avian influenza viruses are segmented RNA viruses classified into subtypes based on their surface glycoproteins, hemagglutinin and neuraminidase. While many low-pathogenic strains exist in wild reservoirs, the highly pathogenic pathotypes, which cause systemic disease and extremely high mortality in domestic poultry, are almost exclusively restricted to the H5 and H7 subtypes.

207. (C) Swollen, purplish snood and intramuscular hemorrhages
Erysipelas in turkeys is an acute, septicemic disease caused by Erysipelothrix rhusiopathiae. The bacterium causes severe vascular damage and congestion throughout the body. Upon post-mortem examination, affected male turkeys typically present with a markedly swollen, turgid, and purplish-to-red snood. This localized lesion is accompanied by widespread hemorrhages in the skeletal muscles and internal organs.

208. (D) Proximal end near the ileocecal junction
The cecal tonsils are the largest masses of lymphoid tissue within the avian intestinal tract. They are located at the proximal end or base of each cecum, where the ceca branch off from the ileum at the ileocecal junction. These lymphoid structures serve a vital role in local mucosal immunity and are frequently examined during necropsies.

209. (C) Pseudomonas aeruginosa
Pseudomonas aeruginosa is an opportunistic, Gram-negative bacterium that can cause acute, localized or systemic infections in poultry. The bacterium produces a characteristic blue-green pigment, pyocyanin, during replication. Systemic infection leads to severe vascular leakage, presenting grossly as green-colored, gelatinous subcutaneous edema, which is frequently accompanied by multifocal areas of hepatic necrosis.

210. (B) Infectious laryngotracheitis
Infectious laryngotracheitis virus is an alphaherpesvirus that causes acute respiratory disease in chickens. The virus replicates in the nuclei of tracheal epithelial cells, inducing a characteristic cytopathic effect. This replication process leads to cell fusion and the formation of multinucleated giant cells, or syncytia, which contain prominent intranuclear inclusion bodies, serving as a pathognomonic diagnostic feature.

****

NODIA

175. (C) Hemorrhagic or necrotic lesions of Peyer's patches and cecal tonsils
Highly virulent, viscerotropic strains of Newcastle disease virus target lymphoid tissues throughout the body. Following systemic infection, the virus localizes within the gut-associated lymphoid tissue, specifically targeting Peyer's patches and the cecal tonsils. This viral replication leads to severe necrotizing and hemorrhagic lesions in these areas, which serve as classic post-mortem diagnostic indicators.

176. (D) Virus isolation followed by RT-PCR and sequencing of the HA cleavage site
Definitive pathotyping of avian influenza viruses is essential for regulatory and diagnostic purposes. While serological tests indicate exposure, pathotyping requires genetic confirmation. This is achieved by isolating the virus in embryonated eggs, followed by RT-PCR amplification and nucleotide sequencing of the hemagglutinin cleavage site to identify the multi-basic amino acid motif characteristic of highly pathogenic strains.

177. (C) Dilated cardiomyopathy causing right or biventricular dilation
Round heart disease is a specific cardiovascular condition that primarily affects young turkey poults. Pathologically, the disease is classified as an idiopathic dilated cardiomyopathy. It is characterized by severe bilateral or right-sided ventricular dilation, resulting in a distinctively rounded and enlarged heart. This cardiac impairment leads to poor circulation, ascites, and sudden death.

178. (B) Wet litter and poor management
Pododermatitis is a common disease of the footpad in broiler flocks, strongly associated with environmental conditions. Wet, poor-quality litter increases the moisture level in contact with the footpad, which softens and compromises the epidermal barrier. This maceration, combined with high ammonia levels, predisposes the footpad to micro-abrasions and subsequent colonization by opportunistic environmental bacteria.

179. (D) Failure of mature cartilage to calcify and undergo vascular invasion, leaving a retained plug
Tibial dyschondroplasia is a skeletal disorder of fast-growing broiler chickens, occurring in the proximal growth plate of the tibia. The condition is characterized by a failure of hypertrophic chondrocytes to undergo normal endochondral ossification. Because this cartilage fails to calcify or become vascularized, a large, non-vascularized plug of retained cartilage persists in the metaphysis, weakening the bone.

180. (A) Trachea
Syngamus trachea, commonly known as the gapeworm, is a nematode parasite that infects the respiratory tract of poultry and wild birds. The adult male and female worms attach to the mucosal lining of the trachea, where they remain in permanent copulation, forming a characteristic Y-shape. This infestation causes severe physical blockage, tracheal irritation, and gasping.

181. (C) Fowl Typhoid
Fowl typhoid is a severe septicemic disease caused by Salmonella Gallinarum. In adult chickens, the acute form leads to significant tissue injury. Upon necropsy, the liver is typically enlarged and mottled, displaying pinpoint white necrotic foci alongside a coppery-bronze discoloration. This is accompanied by severe splenomegaly, which helps differentiate this disease from other septicemic bacterial infections.

182. (A) Dry pox (Fowl Pox)
Dry fowl pox is characterized by proliferative, epithelial lesions on the non-feathered skin of birds, such as the comb and wattles. The virus induces hyperplasia and ballooning degeneration of the cells. As the infection resolves, these raised, crusty, scab-like lesions dry up, desquamate, and peel away, leaving flat, non-elevated, pale scars on the affected tissue.

183. (D) Bacterial entry through the unhealed navel into the yolk sac, causing systemic septicemia
Yolk sac infection, or omphalitis, is a primary cause of mortality in newly hatched chicks. The pathogenesis involves opportunistic bacterial contamination of the unhealed navel shortly after hatching. Bacteria gain entry through this opening, quickly migrating into the nutrient-rich yolk sac where they multiply. This leads to severe, localized inflammation and subsequent systemic septicemia, causing rapid mortality.

184. (B) 3-8 weeks
The bursa of Fabricius is a primary lymphoid organ unique to birds, responsible for the

Downloaded from www.pdf.tube

2007

10. Gross post-mortem examination of an animal that died of chronic starvation typically reveals which characteristic lesion in the coronary groove?
(A) Serous atrophy of fat
(B) Fibrinous epicarditis
(C) Suppurative myocarditis
(D) Extreme lipidosis

2003

11. Consider the following statements regarding cachexia:
I. Mediated by cytokines like TNF-alpha (cachectin).
II. Characterized solely by fat loss with preserved muscle mass.
III. Involves increased basal metabolic rate despite starvation.
Which of the above statements are correct?
(A) I and II
(B) I and III
(C) II and III
(D) I only

2008

12. The gelatinous, translucent appearance of bone marrow in severe protein-energy malnutrition is directly due to the replacement of normal fat with:
(A) Dense fibrous connective tissue
(B) Mucoid extracellular fluid
(C) Extracellular amyloid deposits
(D) Chronic inflammatory exudate

2013

13. Which distinct pathological finding is commonly seen in the lymphoid organs during severe protein-energy malnutrition?
(A) Marked lymphoid atrophy
(B) Follicular hyperplasia
(C) Granulomatous inflammation
(D) Plasmacytosis

2021

14. Which tissue is typically depleted first during the pathogenesis of chronic starvation?
(A) Epicardial fat depot
(B) Bone marrow adipose depot
(C) Subcutaneous fat depot
(D) Perirenal fat depot

2009

15. The profound wasting seen in terminal starvation is largely mediated by extensive catabolism of:
(A) Hepatic glycogen stores
(B) Circulating globulins
(C) Cartilaginous matrix
(D) Skeletal muscle proteins

2017

16. What differentiates kwashiorkor-like syndromes from marasmus-like conditions in veterinary pathology?
(A) Kwashiorkor-like syndromes present with balanced energy and protein starvation.
(B) Marasmus-like conditions typically present with severe hypoproteinemic edema.
(C) Kwashiorkor-like conditions feature selective, severe protein deficiency.
(D) Marasmus-like syndromes are restricted exclusively to mature, adult animals.

2012

17. Which microscopic feature is INCORRECTY associated with serous atrophy of fat?
(A) Adipocytes are shrunken and stellate.
(B) Adipocytes are hyperplastic with large lipid droplets.
(C) The interstitial space contains pale eosinophilic fluid.
(D) The capillary network becomes more prominent.

2025

18. Arrange the following fat depots in the typical order of their mobilization during progressive starvation:
I. Bone marrow fat
II. Subcutaneous fat
III. Omental and perirenal fat
(A) I, II, III
(B) III, II, I
(C) II, I, III
(D) II, III, I

NODIA

2023

19. A deficiency in which group of dietary factors directly precipitates hepatic lipidosis?
(A) Lipotropic factors
(B) Goitrogens
(C) Fat-soluble vitamins
(D) Trace minerals

2009

20. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): Choline deficiency leads to massive accumulation of triglycerides in hepatocytes.
Reason (R): Choline is required for the synthesis of phospholipids, which are essential for

packaging triglycerides into very-low-density lipoproteins (VLDL).
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2023
21. Hypoproteinemic edema, commonly linked to severe protein deficiency, most frequently manifests grossly as:
(A) Ascites and submandibular edema
(B) Diffuse and severe pulmonary hemorrhage
(C) Acute diffuse cerebellar and cerebral edema
(D) Bilateral laryngeal and pharyngeal edema

2005
22. In the context of hepatic lipidosis, what is the primary consequence of decreased apoprotein synthesis?
(A) Accelerated mitochondrial beta-oxidation of fatty acids
(B) Markedly increased hepatocellular glycogen storage
(C) Enhanced rate of parenchymal cellular proliferation
(D) Failure of hepatic triglyceride export

2023
23. Macroscopically, a severely fatty liver (hepatic lipidosis) typically appears:
(A) Shrunken, fibrotic, and nodular
(B) Enlarged, pale yellow, and friable
(C) Dark red with prominent congestion
(D) Small, pale, and gritty

2012
24. Which physiological mechanism explains the development of hypoproteinemic edema?
(A) Increased capillary hydrostatic pressure
(B) Chronic localized lymphatic obstruction
(C) Decreased plasma colloid osmotic pressure
(D) Markedly increased vascular permeability

2014
25. Which of the following is NOT classified as a lipotropic factor?
(A) Active L-methionine
(B) Choline chloride
(C) Cyanocobalamin
(D) Ascorbic acid

2021
26. Identify the INCORRECT pairing of a metabolic imbalance and its consequence.
(A) Severe hypoproteinemia : Low hydrostatic pressure
(B) Lipotropic factor deficiency : Hepating fatty liver syndrome
(C) Severe negative energy balance : Secondary hepatic lipidosis
(D) Chronic severe malnutrition : Serous atrophy of adipose fat

2023
27. Match the specific deficiency or imbalance with its primary pathological manifestation:
List I: (Deficiency or Imbalance)
P. Severe protein deficiency
Q. Choline deficiency
R. Chronic caloric restriction
S. Total starvation
List II: (Pathological Manifestation)
1. Hepatic lipidosis
2. Serous atrophy of fat
3. Hypoproteinemic edema
4. Extreme cachexia
(A) P-3, Q-1, R-4, S-2
(B) P-3, Q-1, R-2, S-4
(C) P-1, Q-3, R-4, S-2
(D) P-2, Q-4, R-1, S-3

2018
28. Which neonatal livestock species is most acutely susceptible to severe hypoglycemia due to low hepatic glycogen reserves at birth?
(A) Calves
(B) Piglets
(C) Foals
(D) Lambs

2003
29. The pathogenesis of neonatal hypoglycemia in piglets is primarily exacerbated by their inability to efficiently perform which metabolic process during the first few days of life?
(A) Gluconeogenesis
(B) Glycolysis
(C) Glycogenolysis
(D) Lipogenesis

2021
30. Consider the following statements regarding neonatal hypoglycemia in livestock:
I. Often triggered by maternal agalactia.
II. Hypothermia accelerates the onset of hypoglycemia.
III. Blood glucose levels remain normal due to

Click Here to Buy book on Amazon

active gluconeogenesis.
IV. Clinical signs include shivering, weakness, and coma.

Which of the above statements are INCORRECT?
(A) II and III
(B) I, III, and IV
(C) III only
(D) I and IV

2013
31. What is the most definitive clinical pathological manifestation of untreated "baby pig disease" (neonatal hypoglycemia)?
(A) Severe tetanic muscle spasms
(B) Generalized severe jaundice
(C) Acute hemorrhagic diarrhea
(D) Encephalopathy and coma

2004
32. In a necropsy of a neonate that died of acute hypoglycemia, the liver typically shows:
(A) Diffuse massive necrosis
(B) Severe amyloidosis deposit
(C) Glycogen depletion
(D) Prominent biliary hyperplasia

2021
33. Maternal agalactia rapidly causes neonatal hypoglycemia because the neonate's brain almost exclusively relies on which substrate for energy?
(A) Ketone bodies
(B) Glucose
(C) Free fatty acids
(D) Amino acids

2023
34. Which mechanism explains why hypothermia significantly worsens neonatal hypoglycemia?
(A) Exposure to cold suppresses systemic insulin secretion entirely.
(B) Low environmental temperatures inhibit renal glucose excretion.
(C) Prolonged hypothermia directly triggers hepatic lipidosis.
(D) Shivering rapidly depletes glucose.

2017
35. Which structural brain lesion is characteristically observed during histopathological examination of neonates with prolonged hypoglycemic coma?
(A) Cortical neuronal degeneration
(B) Diffuse demyelination of the spinal cord
(C) Congenital cerebellar hypoplasia
(D) Focal periventricular hemorrhage

NODIA

2011
36. Which represents the correct sequence of events leading to fatal neonatal hypoglycemia in piglets?
(A) Fasting → Neuroglycopenia → Depletion of hepatic glycogen → Failure of gluconeogenesis
(B) Failure of gluconeogenesis → Fasting → Neuroglycopenia → Depletion of hepatic glycogen
(C) Fasting → Depletion of hepatic glycogen → Failure of gluconeogenesis → Neuroglycopenia
(D) Fasting → Failure of gluconeogenesis → Depletion of hepatic glycogen → Neuroglycopenia

2025
37. The hallmark histological lesion of Vitamin A deficiency across mucosal surfaces is:
(A) Epithelial squamous metaplasia
(B) Hypersecretion of mucus
(C) Mucosal atrophy
(D) Dysplastic intraepithelial neoplasia

2010
38. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): Vitamin A deficiency leads to nyctalopia (night blindness).
Reason (R): 11-cis-retinal is essential for the formation of rhodopsin in the rod cells of the retina.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2024
39. Microphthalmia in calves is a congenital defect frequently linked to maternal deficiency of which nutrient?
(A) Vitamin C
(B) Vitamin A
(C) Vitamin E
(D) Vitamin K

2007
40. In Vitamin A deficiency, occlusion of the parotid salivary duct is primarily caused by:
(A) Impacted calcium carbonate calculi
(B) Chronic granulomatous inflammatory exudate
(C) Keratinized epithelial debris
(D) Multiple constricting fibrous strictures

CHAPTER 12
Nutritional and Production Pathology

2025

1. Which initial mechanism drives cellular swelling during severe nutritional deprivation?
(A) Overactivation of voltage-gated calcium channels
(B) ATP depletion causing failure of the sodium-potassium ATPase pump
(C) Accumulation of intracellular reactive oxygen species
(D) Enhanced function of lysosomal acid hydrolases

2004

2. Under chronic metabolic imbalance, cells undergo a reduction in structural components to maintain survival. Which adaptive process best describes this?
(A) Hyperplasia
(B) Dysplasia
(C) Metaplasia
(D) Atrophy

2011

3. Which statement regarding irreversible cellular injury in nutritional deficiency is INCORRECT?
(A) Intracellular calcium levels strictly remain below normal limits.
(B) It is characterized by severe mitochondrial vacuolization.
(C) It involves profound plasma membrane damage.
(D) It typically culminates in necrosis or apoptosis.

2007

4. Prolonged protein deficiency leads to reduced synthesis of which critical lipid export proteins?
(A) Liposomes
(B) Endosomes
(C) Apoproteins
(D) Lysosomes

2017

5. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): Chronic starvation induces autophagocytosis in parenchymal cells.
Reason (R): Cells degrade their own non-vital organelles to liberate amino acids and energy for essential survival functions.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2023

6. Secondary hypoxia resulting from severe nutritional anemia most profoundly affects which cellular organelle?
(A) Smooth endoplasmic reticulum
(B) Golgi apparatus
(C) Nucleolus
(D) Mitochondria

2007

7. What is the most appropriate term for the replacement of one adult cell type by another due to chronic nutritional stress?
(A) Anaplasia
(B) Metaplasia
(C) Hypertrophy
(D) Aplasia

2023

8. In metabolic imbalances, intracellular accumulation of lipid vacuoles specifically denotes the failure of which cellular process?
(A) Anaerobic glycolysis and lactate production
(B) Ribosomal protein synthesis and translation
(C) Fatty acid oxidation and lipoprotein export
(D) DNA replication and genomic nuclear transcription

2024

9. Which adaptive cellular change is least likely to occur in response to a strict, non-carcinogenic nutritional deficiency?
(A) Atrophy
(B) Metaplasia
(C) Autophagy
(D) Neoplasia

Downloaded from www.pdf.tube

chondrocytes
(D) Absent provisional calcification of cartilage

2009
51. The physiologically active metabolite of Vitamin D that strictly regulates calcium absorption is:
(A) 1,25-dihydroxycholecalciferol
(B) Ergocalciferol
(C) 24,25-dihydroxycholecalciferol
(D) Cholecalciferol

2004
52. Osteomalacia is most commonly observed in which specific animal demographic?
(A) Neonatal puppies during the weaning stage
(B) Fast-growing commercial broiler chicks
(C) Lactating or pregnant adult animals
(D) Rapidly growing thoroughbred foals

2005
53. Which finding is INCORRECT concerning the microscopic appearance of bone in osteomalacia?
(A) The physeal growth plates are markedly thickened.
(B) Osteoid seams are significantly widened.
(C) The proportion of mineralized bone matrix is reduced.
(D) Mature bone trabeculae are lined by excessive unmineralized matrix.

2011
54. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): Endochondral ossification is severely disrupted in rickets.
Reason (R): The lack of mineralization prevents vascular invasion and subsequent replacement of cartilage by bone.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2005
55. White Muscle Disease, caused by a deficiency of Vitamin E and Selenium, primarily affects:
(A) Smooth muscle tissue of viscera
(B) Striated muscle tissue
(C) Smooth and cardiac muscle tissues
(D) Cutaneous skeletal muscle tissue

2018
56. Mulberry Heart Disease, a syndrome characterized by myocardial hemorrhage and necrosis, is primarily seen in which species?
(A) Cattle
(B) Sheep
(C) Swine
(D) Horses

2007
57. Match the Vitamin E/Selenium deficiency syndrome with its target species and primary lesion:
List I: (Syndrome)
P. White Muscle Disease
Q. Exudative Diathesis
R. Mulberry Heart Disease
S. Encephalomalacia
List II: (Target Species and Primary Lesion)
1. Poultry, cerebellar necrosis
2. Swine, microangiopathy and myocardial hemorrhage
3. Calves/Lambs, skeletal myonecrosis
4. Poultry, severe subcutaneous edema
(A) P-1, Q-4, R-2, S-3
(B) P-3, Q-2, R-1, S-4
(C) P-4, Q-3, R-2, S-1
(D) P-3, Q-4, R-2, S-1

NODIA

2012
58. The massive subcutaneous edema observed in chicks with exudative diathesis is due to:
(A) Free radical damage to capillary endothelium
(B) Severe hypoproteinemia and reduced oncotic pressure
(C) Right-sided congestive heart failure
(D) Congenital lymphatic vessel hypoplasia

2005
59. Selenium exerts its antioxidant effect as a critical cofactor for which enzyme?
(A) Superoxide dismutase
(B) Glutathione peroxidase
(C) Catalase
(D) Cytochrome c oxidase

2021
60. The pathological relationship between Vitamin E and Selenium is best described as:
(A) Strictly antagonistic interactions
(B) Synergistic antioxidants
(C) Competitive enzyme inhibition
(D) Mutually exclusive pathways

2013
61. Which histopathological feature is NOT characteristic of skeletal muscle in White Muscle Disease?
(A) Massive eosinophilic infiltration
(B) Hyaline degeneration of myofibers
(C) Mineralization of necrotic fibers
(D) Macrophage infiltration

2010
62. The underlying pathogenesis of the lesions in Mulberry Heart Disease primarily involves:
(A) Direct viral-mediated lysis of cardiomyocytes
(B) Excessive lipid deposition in the myocardium
(C) Microangiopathy and thrombosis
(D) Autoimmune-mediated destruction of valves

2010
63. Consider the following diseases:
I. White Muscle Disease
II. Exudative Diathesis
III. Swayback
Which of the above diseases are correctly attributed to Vitamin E and Selenium deficiency?
(A) II and III
(B) I and III
(C) I, II, and III
(D) I and II

2010
64. Polioencephalomalacia in ruminants is primarily associated with a deficiency of:
(A) Riboflavin
(B) Pyridoxine
(C) Thiamine
(D) Cyanocobalamin

2016
65. Given below are two statements, one is labelled as Assertion (A) and the other is labelled as Reason (R).
Assertion (A): Ingestion of plants like bracken fern can induce cerebrocortical necrosis in livestock.
Reason (R): Bracken fern contains thiaminases that destroy thiamine, leading to neuronal energy failure.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2017
66. "Curled-toe paralysis" in chicks is the classic pathognomonic sign of which deficiency?
(A) Thiamine
(B) Pantothenic acid
(C) Niacin
(D) Riboflavin

2019
67. The neurological lesion responsible for curled-toe paralysis is:
(A) Sciatic nerve Schwann cell hyperplasia
(B) Diffuse demyelination of the brainstem nuclei
(C) Acute localized motor cortex necrosis
(D) Degeneration of spinal cord white tracts

2019
68. Systemic polyneuritis with prominent peripheral nerve demyelination in poultry is typically a manifestation of:
(A) Biotin deficiency
(B) Vitamin C deficiency
(C) Thiamine deficiency
(D) Vitamin E deficiency

2004
69. Gross examination of the brain in acute polioencephalomalacia often reveals which unique characteristic under ultraviolet light?
(A) Necrotic cerebral cortex fluorescence
(B) Prominate punctate red cortical fluorescence
(C) Bright yellow autofluorescence of meninges
(D) Complete and absolute absence of fluorescence

2019
70. Which microscopic finding is INCORRECT for polioencephalomalacia?
(A) Diffuse spongiosis of the cerebral cortex
(B) Necrotic neurons with pyknotic nuclei
(C) Prominent gitter cell accumulation in late stages
(D) Cerebellar peduncle demyelination

2013
71. Which condition is strictly NOT a manifestation of a Vitamin B-complex deficiency?
(A) Curled-toe paralysis
(B) Enzootic ataxia
(C) Black tongue in dogs
(D) Polioencephalomalacia

2015
72. Match the Vitamin B-complex member with its corresponding deficiency disease:
List I: (Vitamin B-complex Member)

Click Here to Buy book on Flipkart

P. Thiamine (B1)
Q. Riboflavin (B2)
R. Niacin (B3)
S. Cyanocobalamin (B12)

List II: (Deficiency Disease)

1. Curled-toe paralysis
2. Polioencephalomalacia
3. Wasting secondary to Cobalt deficiency
4. Pellagra/Black tongue

(A) P-1, Q-2, R-3, S-4
(B) P-3, Q-1, R-4, S-2
(C) P-2, Q-1, R-4, S-3
(D) P-2, Q-4, R-1, S-3

2025

73. Vitamin K is essential for the hepatic synthesis of which specific set of coagulation factors?
(A) I, II, V, X
(B) VIII, IX, XI, XII
(C) III, IV, V, VII
(D) II, VII, IX, X

2016

74. Consider the following statements regarding Vitamin K antagonism:
I. Dicoumarol is a potent Vitamin K antagonist.
II. It leads to profound hemorrhagic diathesis.
III. It inhibits the enzyme vitamin K epoxide reductase.
IV. It causes massive intravascular thrombosis.
Which of the above statements are correct?
(A) I, II, and III
(B) II, III, and IV
(C) I, III, and IV
(D) I and IV

2007

75. In cattle, severe fatal hemorrhage resulting from Vitamin K antagonism is most classically associated with ingestion of:
(A) Bracken fern
(B) Moldy sweet clover
(C) Lupine species
(D) Locoweed

2022

76. The molecular consequence of inhibiting vitamin K epoxide reductase is:
(A) Rapid degradation of all circulating platelets
(B) Inability to recycle oxidized vitamin K
(C) Direct chemical lysis of vascular endothelial cells
(D) Overproduction of endogenous plasminogen activator

2003

77. The primary clinical pathological manifestation of severe Vitamin K deficiency or antagonism is:
(A) Generalized hemorrhagic diathesis
(B) Petechiae restricted to mucous membranes
(C) Microangiopathic hemolytic anemia
(D) Purulent lymphadenitis of regional nodes

2018

78. Vitamin K facilitates blood coagulation by acting as a cofactor for the:
(A) Alpha-hydroxylation of polypeptide proline
(B) Mitochondrial beta-oxidation of fatty acids
(C) Gamma-carboxylation of glutamic acid
(D) Enzymatic methylation of cellular homocysteine

2005

79. Which statement INCORRECTLY pairs a clotting factor with its behavior in Vitamin K antagonism?
(A) Factor VII depletes rapidly due to its short half-life, prolonging PT initially.
(B) Activated partial thromboplastin time (aPTT) also becomes prolonged as Factors IX and X deplete.
(C) Inactive precursor forms (PIVKAs) accumulate in the blood.
(D) Factor II (Prothrombin) has the shortest half-life and depletes first.

2017

80. Second-generation anticoagulant rodenticides (e.g., brodifacoum) cause toxicity via which mechanism?
(A) Severe acute hepatotoxicity and cellular necrosis
(B) Direct immunological destruction of megakaryocytes
(C) Inhibition of vitamin K epoxide reductase
(D) Competitive binding of anticoagulant to fibrinogen

2025

81. Which of the following is NOT characteristically observed in an animal with sweet clover poisoning?
(A) Large intramuscular hematomas
(B) Thrombocytosis
(C) Severe bilateral hemarthrosis
(D) Profuse gastrointestinal melena

2009

82. Nutritional secondary hyperparathyroidism is classically induced by a diet that is:
(A) High Calcium, Low Phosphorus

Downloaded from www.pdf.tube

hepatic tissue
(D) Glucagon : Stimulates gluconeogenesis in liver cells

2003
125. Which of the following conditions is NOT classified as a periparturient production disease in dairy cattle?
(A) Bovine viral diarrhea
(B) Milk fever
(C) Ketosis
(D) Post-parturient haemoglobinuria

2017
126. Acute hypocalcemia (Milk fever) in cows classically manifests as:
(A) Rigid tetany and convulsions
(B) Intention tremors
(C) Flaccid paralysis
(D) Ascending myelomalacia

2023
127. At the neuromuscular junction, what is the specific role of calcium that fails during milk fever?
(A) It mediates direct influx of sodium ions into muscle fibers.
(B) It degrades acetylcholine within the synaptic cleft.
(C) It prevents repolarization of the presynaptic nerve terminal.
(D) It triggers synaptic vesicle fusion and acetylcholine release.

2008
128. Consider the following clinical stages of milk fever (parturient paresis) in cows:
I. Sternal recumbency with an "S-shaped" curve of the neck
II. Lateral recumbency, coma, and potential death
III. Brief period of excitement, muscle tremors, and ataxia
Which of the following represents the correct chronological sequence of these stages?
(A) I, II, III
(B) III, I, II
(C) II, I, III
(D) I, III, II

2015
129. The flaccid paralysis seen in bovine milk fever occurs because low extracellular calcium leads to:
(A) Continuous and unregulated firing of the motor neuron
(B) Failure of acetylcholine release at the endplate
(C) Irreversible binding of calcium ions to troponin C
(D) Rapid destruction of active motor endplate receptors

2007
130. A pathognomonic clinical posture of a cow in Stage II of milk fever is:
(A) Rigid sawhorse standing posture with stiffened limbs
(B) Pronounced opisthotonos with bilateral forelimb extension
(C) Persistent dog-sitting posture with hindlimb weakness
(D) Sternal recumbency with the head tucked into the flank

2007
131. In the context of milk fever pathophysiology, which fraction of blood calcium is biologically critical and acutely depleted?
(A) Ionized calcium
(B) Protein-bound calcium
(C) Complexed calcium
(D) Intracellular calcium

NODIA

2021
132. Hypocalcemia exacerbates its own severity by causing paralysis of which specific tissue, leading to a vicious cycle of decreased calcium absorption?
(A) Somatic skeletal muscle
(B) Myocardial cardiac muscle
(C) Gastrointestinal smooth muscle
(D) Diaphragmatic respiratory muscle

2021
133. Which statement INCORRECTLY details the pathophysiology of parturient paresis?
(A) It involves a primary defect in parathyroid gland structure.
(B) It coincides with the sudden onset of copious milk production.
(C) Fecal and urinary calcium losses exceed intestinal absorption.
(D) High magnesium prevents synaptic release of acetylcholine.

2005
134. Which factor LEAST contributes to the onset of milk fever in a high-yielding dairy cow?
(A) Age of the cow (older cows are more susceptible)
(B) High dietary calcium intake during the dry

(A) Mitochondrial lysyl oxidase
(B) Cytochrome c oxidase
(C) Melanogenic tyrosinase
(D) Superoxide dismutase

2012
93. Achromotrichia (loss of hair pigmentation) in copper deficiency results from the impairment of:
(A) Tyrosinase
(B) Ceruloplasmin
(C) Dopamine beta-hydroxylase
(D) Ascorbic acid oxidase

2011
94. Spontaneous cardiovascular ruptures in copper-deficient animals are caused by defective cross-linking of collagen and elastin, a process mediated by:
(A) Matrix metalloproteinases
(B) Prolyl hydroxylase
(C) Lysyl oxidase
(D) Elastase

2012
95. The characteristic gross brain lesion observed in severe congenital swayback is:
(A) Diffuse cerebellar cortical hypoplasia
(B) Severe hemorrhage in the basal ganglia
(C) Acute suppurative meningoencephalitis
(D) Cerebral white matter cavitation

2022
96. Which microscopic feature is NOT observed in the spinal cord of lambs with enzootic ataxia?
(A) Degeneration of motor neurons in the ventral horn
(B) Demyelination of descending motor tracts
(C) Massive neutrophil infiltration
(D) Wallerian-type degeneration

2014
97. Secondary copper deficiency in ruminants is most commonly induced by excess dietary intake of:
(A) Iron and Zinc
(B) Molybdenum and Sulfur
(C) Calcium and Phosphorus
(D) Selenium and Vitamin E

2012
98. Which of the following is NOT a copper-dependent metalloenzyme?
(A) Serum ceruloplasmin
(B) Mitochondrial cytochrome c oxidase
(C) Extracellular lysyl oxidase
(D) Glutathione peroxidase

2008
99. The primary etiology of piglet anemia is the rapid growth of neonates combined with:
(A) Low iron in sow's milk and no soil access
(B) Severe umbilical hemorrhage during birth
(C) Autoimmune destruction of neonatal red blood cells
(D) Inability to absorb intestinal iron due to achlorhydria

2006
100. Consider the following statements regarding iron deficiency anemia in piglets:
I. It presents as a microcytic, hypochromic anemia.
II. It is characterized by polycythemia.
III. Clinical signs include pale mucous membranes, lethargy, and dyspnea ("thumps").
Which of the above statements are correct?
(A) I and II
(B) II and III
(C) I and III
(D) I, II, and III

2002
101. Profound wasting disease (pine) in ruminants is directly caused by a deficiency of:
(A) Cobalt
(B) Iron
(C) Copper
(D) Zinc

2025
102. Cobalt is an essential structural component of which crucial ruminant vitamin?
(A) Thiamine pyrophosphate
(B) Active tetrahydrofolic acid
(C) L-ascorbic acid
(D) Cyanocobalamin

2001
103. The characteristic appearance of erythrocytes in a peripheral blood smear of a piglet with severe iron deficiency is:
(A) Large size with basophilic stippling
(B) Small size and pale central pallor
(C) Spherical shape lacking central pallor
(D) Sickle-shaped with prominent Heinz bodies

2012
104. "White liver disease" in sheep, characterized by severe hepatic lipidosis, is a known sequela of:
(A) Chronic copper toxicity
(B) Systemic iron overload
(C) Cobalt deficiency
(D) Dietary zinc deficiency

Click Here to Buy book on Amazon

(B) Low Calcium, Low Phosphorus
(C) High Magnesium, Low Calcium
(D) High Phosphorus, Low Calcium

2008

83. "Bran disease," characterized by severe facial bone distortion, is a form of nutritional secondary hyperparathyroidism traditionally seen in:
(A) Horses
(B) Pigs
(C) Cattle
(D) Dogs

2021

84. Match the disease terminology with its underlying pathology related to calcium or phosphorus imbalance:
List I: (Disease Terminology)
P. Bran disease
Q. Rubber jaw syndrome
R. Big head disease
S. Rickets
List II: (Underlying Pathology)
1. Dogs with severe renal or nutritional secondary hyperparathyroidism
2. Horses with facial osteodystrophia fibrosa
3. Defective mineralization in young animals
4. Goats/Sheep with severe facial bone swelling
(A) P-1, Q-2, R-3, S-4
(B) P-3, Q-1, R-2, S-4
(C) P-2, Q-1, R-4, S-3
(D) P-2, Q-3, R-1, S-4

2006

85. The direct cause of parathyroid gland hyperplasia in nutritional secondary hyperparathyroidism is:
(A) Chronic hypocalcemia
(B) Elevated serum phosphorus
(C) Chronic hypercalcemia
(D) Excessive Vitamin D intake

2005

86. The hallmark microscopic lesion of osteodystrophia fibrosa is:
(A) Diffuse massive necrosis of mature osteocytes
(B) Severe accumulation of unmineralized osteoid
(C) Granulomatous inflammation of the marrow cavity
(D) Fibrous tissue replacement of bone

2023

87. Excessive resorption of cancellous bone of the maxilla and mandible, leading to bilateral facial swelling, is characteristic of:
(A) Congenital osteopetrosis
(B) Osteodystrophia fibrosa
(C) Systemic osteochondrosis
(D) Purulent osteomyelitis

2015

88. In the pathogenesis of osteodystrophia fibrosa, the action of parathyroid hormone specifically stimulates:
(A) Osteoblastic bone deposition
(B) Chondrocyte proliferation
(C) Osteoclastic bone resorption
(D) Fibroblast apoptosis

2003

89. Which feature distinguishes nutritional secondary hyperparathyroidism from primary hyperparathyroidism?
(A) Extent and severity of cortical bone resorption
(B) Diffuse fibrous tissue proliferation in bone
(C) Presence and activity of multinucleated osteoclasts
(D) Diffuse hyperplasia vs autonomous adenoma

2002

90. Consider the following statements regarding the pathogenesis of nutritional secondary hyperparathyroidism:
I. High dietary phosphorus binds intestinal calcium.
II. Serum calcium levels fall, stimulating PTH release.
III. PTH promotes renal excretion of calcium.
IV. PTH stimulates osteoclasts to resorb bone.
V. Chronic stimulation leads to fibrous osteodystrophy.
Which of the above statements is/are INCORRECT?
(A) III only
(B) I, II, and IV
(C) II, IV, and V
(D) I, III, and V

2007

91. Copper deficiency in pregnant ewes typically results in which neurological condition in their lambs?
(A) Transmissible scrapie
(B) Enzootic ataxia
(C) Congenital border disease
(D) Acute polioencephalomalacia

2012

92. The demyelinating lesions of swayback are attributed to the functional failure of which copper-dependent enzyme?

NODIA

(D) Calcium

2002

115. Which clinical sign is NOT typically associated with iodine deficiency in livestock?
(A) Severe exophthalmos
(B) Prolonged gestation
(C) Myxedema
(D) Birth of weak or stillborn offspring

2024

116. Which feature is LEAST relevant to zinc deficiency pathology?
(A) Crusty skin lesions on the face and distal limbs
(B) Impaired wound healing
(C) Severe hypochromic macrocytic anemia
(D) Thymic hypoplasia

2011

117. In veterinary medicine, the term "production disease" refers primarily to metabolic breakdowns caused by:
(A) High production demands exceeding animal homeostatic capacity
(B) Pathogenic infectious agents endemic in high-density facilities
(C) Inherited genetic mutations that result in developmental failure
(D) Chronic exposure to hazardous chemical pollutants in the environment

2014

118. Which physiological control system prioritizes the mobilization of maternal tissues to support lactation, often at the expense of the dam's overall health?
(A) Homeostasis
(B) Feedback inhibition
(C) Allostasis
(D) Homeorhesis

2010

119. Consider the following statements regarding the metabolic stress factors defining production diseases in high-yielding dairy animals:
I. Extreme genetic selection for milk yield alters energy partitioning.
II. Post-partum dry matter intake often lags behind energy output.
III. Negative energy balance triggers massive fat mobilization.
IV. The incidence of production diseases peaks during mid-lactation.
V. Hypocalcemia and ketosis are classic examples.
Which of the above statements are correct?
(A) I, III, IV, and V
(B) I, II, III, and V
(C) II, III, IV, and V
(D) I, II, IV, and V

2010

120. Negative energy balance in early lactation drives the massive mobilization of:
(A) Structural proteins derived from skeletal muscle fibers
(B) Intracellular calcium reserves stored within cortical bone
(C) Non-esterified fatty acids mobilized from adipose tissues
(D) Labile iron stores sequestered within hepatic hepatocytes

2015

121. The most critical indicator of homeostatic failure in periparturient dairy cattle is:
(A) Progressive increases in maternal body condition scores
(B) Transient hyperglycemia during the immediate post-partum
(C) Marked decreases in resting heart rate and cardiac output
(D) Dynamic alterations in serum metabolite and mineral profiles

2016

122. The periparturient immunosuppression frequently seen in production diseases is largely mediated by elevated circulating levels of:
(A) Cortisol
(B) Oxytocin
(C) Insulin
(D) Glucagon

2007

123. The root cause of almost all periparturient production diseases in dairy cattle is the sudden, enormous diversion of nutrients to the:
(A) Developing fetus
(B) Mammary gland
(C) Rumen compartment
(D) Uterine wall

2002

124. Identify the INCORRECT pair regarding metabolic adaptations in high-yielding cows.
(A) Somatotropin : Stimulates lipolysis and milk synthesis
(B) Insulin : Elevates during early lactation to conserve fat
(C) Epinephrine : Promotes glycogenolysis in

Downloaded from www.pdf.tube

2002

105. Ruminants rely heavily on Vitamin B12 for the activity of methylmalonyl-CoA mutase. Failure of this enzyme directly disrupts the metabolism of:
(A) Acetate ions
(B) Butyrate ions
(C) Lactate ions
(D) Propionate

2014

106. Which statement regarding iron metabolism is INCORRECT?
(A) Hepcidin upregulates iron absorption during deficiency.
(B) Iron is transported in the blood bound to transferrin.
(C) Iron is stored intracellularly as ferritin or hemosiderin.
(D) Piglets have highly limited total body iron stores at birth.

2021

107. Which represents the correct sequence of events leading to clinical piglet anemia?
(A) Reduced hemoglobin synthesis → Lack of dietary iron → Depletion of iron stores → Microcytic hypochromic anemia
(B) Lack of dietary iron → Depletion of iron stores → Reduced hemoglobin synthesis → Microcytic hypochromic anemia
(C) Microcytic hypochromic anemia → Lack of dietary iron → Depletion of iron stores → Reduced hemoglobin synthesis
(D) Lack of dietary iron → Microcytic hypochromic anemia → Reduced hemoglobin synthesis → Depletion of iron stores

2001

108. The classic dermatological lesion associated with zinc deficiency in domestic animals is:
(A) Epidermal orthokeratosis
(B) Epidermal parakeratosis
(C) Vesiculopustular dermatitis
(D) Nodular granulomatous dermatitis

2002

109. Histologically, epidermal parakeratosis is defined as:
(A) Retention of nuclei in the stratum corneum
(B) Diffuse thickening of the stratum spinosum layer
(C) Complete loss of cells within the stratum basale
(D) Infiltration of active eosinophils in the dermis

2006

110. Consider the following statements regarding iodine deficiency:
I. It results in a hyperplastic goiter.
II. It leads to increased production of thyroxine (T4).
III. Neonates born to deficient mothers may be alopecic and weak.
IV. It causes suppression of Thyroid Stimulating Hormone (TSH).
Which of the above statements are INCORRECT?
(A) I and III
(B) I and IV
(C) II and IV
(D) II and III

2023

111. The enlargement of the thyroid gland (goiter) in iodine deficiency is a direct compensatory response driven by elevated levels of:
(A) Secreted thyroglobulin protein
(B) Thyroidal calcitonin hormone
(C) Circulating parathyroid hormone
(D) Thyroid-stimulating hormone

2025

112. Microscopic examination of a hyperplastic goiter due to iodine deficiency reveals:
(A) Large, distended follicles filled with dense, eosinophilic colloid
(B) Massive lymphocytic infiltration leading to follicular destruction
(C) Follicles lined by tall columnar cells with minimal colloid
(D) Neoplastic proliferation of thyroid parafollicular C-cells

NODIA

2009

113. Goitrogenic compounds, such as those found in Brassica plants, induce goiter by:
(A) Directly destroying pituitary cells secreting trophic hormones
(B) Inhibiting iodine uptake or organic binding by thyroid tissue
(C) Binding directly to and blocking active pituitary TSH receptors
(D) Promoting autoantibody production directed against thyroglobulin

2021

114. Swine fed diets excessively high in which mineral are predisposed to developing secondary zinc deficiency parakeratosis?
(A) Phosphorus
(B) Copper
(C) Iron

period
(C) High dietary cation-anion difference (DCAD) diets pre-partum
(D) High dietary magnesium intake pre-partum

2002
135. The typical clinico-pathological blood profile of a cow experiencing acute milk fever reveals:
(A) Hypocalcemia, hyperphosphatemia, and hypomagnesemia
(B) Hypocalcemia, hypophosphatemia, and hypermagnesemia
(C) Hypercalcemia, hypophosphatemia, and hypermagnesemia
(D) Hypocalcemia, normal phosphate, and hypomagnesemia

2011
136. A significant contributing factor to milk fever is the status of the parathyroid gland just prior to parturition in cows fed high calcium diets, which is typically:
(A) Hyperactive and hypertrophied
(B) Undergoing neoplastic transformation
(C) Sluggish and unresponsive
(D) Acutely inflamed

2003
137. Prolonged recumbency in milk fever ("downer cow syndrome") frequently leads to which severe secondary complication?
(A) Secondary cerebellar herniation
(B) Acute left-displaced abomasum
(C) Retained placental membranes
(D) Hindlimb ischemic crush myopathy

2003
138. Aspiration pneumonia is a common fatal complication of milk fever due to:
(A) Loss of the swallowing reflex and silent regurgitation
(B) Severe systemic immunosuppression mediated by high cortisol
(C) Direct viral invasion of the alveolar pulmonary epithelium
(D) Secondary opportunistic proliferation of Pasteurella multocida

2013
139. Ischemic muscle necrosis in a downer cow is primarily initiated by:
(A) Direct toxicity of low intracellular calcium on myocytes
(B) Local compression of vasculature by high pressure
(C) Prolonged episodes of severe systemic hypotension
(D) Acute bacterial myositis of the pelvic limbs

2005
140. Which serum biochemical marker provides the most definitive evidence of crush myopathy in a cow recovering from prolonged milk fever?
(A) Alkaline phosphatase (ALP)
(B) Creatine kinase (CK)
(C) Gamma-glutamyl transferase (GGT)
(D) Blood urea nitrogen (BUN)

2017
141. Identify the INCORRECT pairing between a milk fever complication and its underlying physiological cause.
(A) Mastitis : Complete failure of milk synthesis
(B) Uterine prolapse : Uterine smooth muscle atony
(C) Bloat : Rumen stasis and failure of eructation
(D) Aspiration pneumonia : Pharyngeal paralysis

2003
142. A cow with a blood calcium level of 4.5 mg/dL (normal 8.5-10.4 mg/dL) will most likely exhibit:
(A) Aggression and hyperesthesia
(B) Normal gait but reduced milk yield
(C) Coma or severe flaccid paresis
(D) Mild muscle fasciculations

2010
143. Which of the following conditions is LEAST likely to be a direct secondary complication of milk fever?
(A) Coliform mastitis
(B) Dystocia
(C) Retained fetal membranes
(D) Hepating lipidosis

2016
144. The fundamental pathogenesis of ketosis (acetonemia) in high-yielding dairy cattle begins with:
(A) Excessive carbohydrate intake
(B) Primary pancreatic beta-cell failure
(C) Severe negative energy balance
(D) Defective lipid absorption

2002
145. In bovine ketosis, the heavy demand for glucose for milk lactose synthesis severely depletes which critical Krebs cycle intermediate?
(A) Citrate
(B) Oxaloacetate
(C) Alpha-ketoglutarate
(D) Succinate

Click Here to Buy book on Flipkart

2005
146. Gluconeogenesis in the ruminant liver is overwhelmingly dependent on the supply of which volatile fatty acid from the rumen?
(A) Acetate
(B) Butyrate
(C) Valerate
(D) Propionate

2005
147. How is hepatic lipidosis mechanistically linked to clinical ketosis in cattle?
(A) NEFA influx exceeds liver oxidation, causing ketogenesis and fat storage.
(B) Ketones directly destroy VLDL transport particles, trapping lipid within the liver.
(C) Hepatic lipidosis completely blocks further entry of circulating NEFAs into the liver.
(D) Ketosis increases insulin levels, which actively drives downstream lipid deposition.

2001
148. The major ketone bodies accumulated in the blood of cows with acetonemia include:
(A) Pyruvate, lactate, and oxaloacetate intermediates
(B) Alpha-ketoglutarate, malate, and citrate molecules
(C) Acetoacetate, beta-hydroxybutyrate, and acetone
(D) Acetyl-CoA, malonyl-CoA, and propionyl-CoA esters

2025
149. The clinical syndrome of "nervous ketosis" is believed to result directly from:
(A) Direct neurotoxic effects of high ketones paired with neuroglycopenia
(B) Acute cerebral anoxia resulting from severe alveolar hypoventilation
(C) Traumatic intracranial hemorrhage within the cerebrovascular system
(D) Subclinical hypomagnesemic tetany occurring concurrently in the herd

2018
150. Which statement INCORRECTLY details the biochemical dysfunction in ketosis?
(A) Lack of oxaloacetate stalls the tricarboxylic acid (TCA) cycle.
(B) Acetyl-CoA cannot condense with oxaloacetate to form citrate.
(C) Excess Acetyl-CoA is shunted into ketogenesis.
(D) Excess Acetyl-CoA is converted entirely to glucose.

2007
151. Which substrate is NOT a precursor for gluconeogenesis in the bovine liver?
(A) Propionate
(B) Acetate
(C) Glycerol
(D) Glucogenic amino acids

2010
152. The characteristic "sweet or fruity" breath odor in cows with ketosis is due to pulmonary excretion of:
(A) Acetoacetate
(B) Beta-hydroxybutyrate
(C) Acetone
(D) Isopropanol

2024
153. Pregnancy toxemia (ovine ketosis) is most heavily precipitated by the severe energy demands of:
(A) Early embryonic development and initial implantation within the uterus
(B) Mid-gestation placental growth and vascularization of the cotyledons
(C) High-volume colostrum production in the mammary gland post-lambing
(D) Rapid late-gestation fetal growth, especially with multiple fetuses

2023
154. The neurological signs of pregnancy toxemia in sheep, such as blindness and star-gazing, are primarily caused by:
(A) Neuroglycopenia
(B) Cerebellar hypoplasia
(C) Thiamine deficiency
(D) Primary viral encephalitis

NODIA

2024
155. Why are pregnant ewes carrying twins particularly susceptible to pregnancy toxemia?
(A) Enlarged twins compress the maternal liver, directly impairing fatty acid metabolism.
(B) Combined fetal glucose demands exceed the ewe's maximal gluconeogenic capacity.
(C) Multiple fetuses secrete specific metabolic toxins that inhibit gluconeogenesis.
(D) The ewe develops a severe, systemic autoimmune reaction to twin fetal proteins.

2021
156. The most prominent gross lesion in the liver of a sheep that died of pregnancy toxemia is:
(A) A shrunken, fibrotic, and multinodular hepatic parenchyma
(B) A pale yellow, enlarged, greasy, and friable

liver
(C) A diffuse dark greenish-black liver with multiple cystic cavities
(D) Multifocal, well-circumscribed purulent abscesses throughout all lobes

2009
157. Histologically, the encephalopathy associated with pregnancy toxemia is characterized by:
(A) Astrocyte swelling and neuronal necrosis
(B) Massive neutrophil infiltration of the brain
(C) Severe demyelination of the cervical spinal cord
(D) Diffuse microglial nodule formation in the cortex

2002
158. The fundamental physiological difference between the onset of ketosis in cattle vs. sheep is:
(A) Cattle develop ketosis during the pre-partum stage, whereas sheep develop it post-partum.
(B) Sheep ketosis arises as a consequence of a primary systemic failure of lipolysis.
(C) Cattle ketosis is driven by lactation, whereas sheep ketosis is driven by fetal growth.
(D) Cattle ketosis biochemical pathways utilize only acetoacetate as a fuel source.

2023
159. Which hormonal change is INCORRECTLY associated with the pathogenesis of pregnancy toxemia?
(A) Elevation of maternal cortisol levels
(B) Decreased insulin levels to spare glucose for the fetus
(C) Elevation of glucagon
(D) Massive increases in circulating insulin

2006
160. In the terminal stages of pregnancy toxemia, ewes often develop renal failure due to dehydration and shock, indicated by elevated levels of:
(A) Serum glucose
(B) Serum calcium
(C) BUN and creatinine
(D) Conjugated bilirubin

2002
161. Which finding is LEAST likely to be found on necropsy of an ewe that died of pregnancy toxemia?
(A) Pale, enlarged, friable liver
(B) Massive fibrinopurulent peritonitis
(C) Multiple large, late-term fetuses in utero
(D) Empty or minimally filled rumen

2019
162. Magnesium tetany (Grass staggers) results from an acute hypomagnesemia which critically alters neuromuscular irritability. At the normal neuromuscular junction, magnesium acts to:
(A) Upregulate acetylcholine synthesis within the presynaptic terminal
(B) Irreversibly bind and block postsynaptic acetylcholine receptors
(C) Block voltage-gated sodium channels directly on the muscle membrane
(D) Competitively antagonize calcium to regulate acetylcholine release

2010
163. The onset of clinical tetany in grass staggers correlates most closely with severely depressed magnesium concentrations in which biological fluid?
(A) Cerebrospinal fluid
(B) Circulating blood serum
(C) Excreted voided urine
(D) Intracellular cytosol

2014
164. Acute hypomagnesemia in cattle grazing lush spring pastures is heavily exacerbated by high dietary levels of:
(A) Sodium
(B) Calcium and Phosphorus
(C) Potassium and Nitrogen
(D) Copper and Zinc

2013
165. The tetanic muscle spasms in grass staggers occur because critically low magnesium levels lead to:
(A) Excessive release of acetylcholine at the motor endplate
(B) Complete failure of acetylcholine release from the axon terminal
(C) Complete blockade of active postsynaptic receptors at the synapse
(D) Severe flaccid paralysis of the presynaptic nerve membrane

NODIA

2006
166. In a suspected case of sudden death due to grass staggers, the most reliable post-mortem fluid sample for detecting hypomagnesemia is:
(A) Circulating whole blood
(B) Harvested pericardial fluid
(C) Urinary bladder content
(D) Vitreous humor or CSF

Subcutaneous adipose tissue is mobilized first to meet immediate deficits, followed by the internal visceral depots, specifically omental and perirenal fat. The bone marrow fat represents a critical, deep reserve that is mobilized last during terminal starvation.

19. (A) Lipotropic factors
A dietary deficiency in lipotropic factors, such as choline and methionine, directly prevents the normal packaging and export of lipids from hepatocytes. These essential factors are critical for the biosynthesis of phosphatidylcholine, a major constituent of very-low-density lipoproteins. Without lipotropic factors, triglycerides cannot be exported, causing severe accumulation and initiating hepatic lipidosis.

20. (A) Both A and R are true and R is the correct explanation of A.
Choline is a critical lipotropic nutrient required for the synthesis of phosphatidylcholine, a key structural phospholipid. Phospholipids are indispensable for assembling the outer membrane of very-low-density lipoproteins, which transport triglycerides out of hepatocytes. A deficiency in choline directly impairs this packaging mechanism, preventing lipid export and resulting in massive accumulation of triglycerides.

21. (A) Ascites and submandibular edema
Severe dietary protein deficiency results in extreme hypoproteinemia, primarily due to decreased albumin synthesis by the liver. The loss of intravascular albumin significantly lowers plasma colloid osmotic pressure, allowing fluid to escape capillaries into interstitial spaces and body cavities. This manifests grossly as dependent subcutaneous edema, particularly in the submandibular region, and ascites.

22. (D) Failure of hepatic triglyceride export
Apoproteins are vital protein components synthesized by hepatocytes to bind with lipids, forming water-soluble lipoprotein complexes. When apoprotein synthesis is compromised, hepatocytes cannot package accumulated triglycerides into very-low-density lipoproteins. Consequently, these lipid molecules cannot be exported into the circulation, resulting in their physical retention inside the hepatocellular cytoplasm and severe lipidosis.

23. (B) Enlarged, pale yellow, and friable
A liver with severe hepatic lipidosis undergoes dramatic macromorphological changes due to the massive accumulation of intracellular lipid vacuoles. Grossly, the liver becomes markedly enlarged with rounded margins, exhibits a pale yellow or clay-colored appearance, and feels soft or friable to the touch. In water, sections of the tissue may float due to lipid content.

24. (C) Decreased plasma colloid osmotic pressure
The development of hypoproteinemic edema is driven by a reduction in plasma colloid osmotic pressure, which is primarily maintained by circulating albumin. When albumin levels fall due to starvation or protein loss, the osmotic force pulling fluid back into capillaries is diminished. Consequently, fluid leaks into the interstitial tissues, leading to generalized edema and ascites.

25. (D) Ascorbic acid
Lipotropic factors are specific compounds, including choline, methionine, and cyanocobalamin, that facilitate the synthesis, packaging, and export of lipids from hepatocytes. These substances prevent the abnormal accumulation of fat within the liver. Ascorbic acid, also known as Vitamin C, acts as a water-soluble antioxidant and cofactor but is not classified as a lipotropic agent.

26. (A) Severe hypoproteinemia : Low hydrostatic pressure
Severe hypoproteinemia is characterized by a significant decrease in plasma colloid osmotic pressure rather than a decrease in hydrostatic pressure. This low osmotic pressure disrupts the Starling forces, preventing fluid resorption at the venous end of capillaries and driving edema. The other options represent accurate pairings of nutritional and metabolic imbalances with their recognized pathological outcomes.

27. (B) P-3, Q-1, R-2, S-4
Severe protein deficiency causes hypoproteinemic edema due to diminished albumin synthesis. Choline deficiency, a lipotropic defect, causes hepatic lipidosis by preventing very-low-density lipoprotein assembly. Chronic caloric restriction leads to serous atrophy of fat as adipose tissue is progressively depleted. Finally, total starvation culminates in extreme cachexia with extensive catabolism of both fat and muscle reserves.

Downloaded from www.pdf.tube

2002
178. Following ruminal mucosal damage from acidosis, bacteria are transported to the liver via the portal circulation. The principal bacterium involved in the subsequent liver abscesses is:
(A) Clostridium chauvoei
(B) Trueperella pyogenes
(C) Escherichia coli
(D) Fusobacterium necrophorum

2008
179. A fatal complication of the rumenitis-liver abscess complex is the extension of infection from a hepatic abscess into the:
(A) Caudal vena cava, causing thrombosis
(B) Gallbladder, causing cholecystitis
(C) Portal vein, causing portal hypertension
(D) Hepatic artery, causing aneurysm

2001
180. The underlying cause of chemical rumenitis in the sequelae of grain overload is:
(A) Massive systemic secretion of bacterial endotoxins
(B) Excessive accumulation of free ruminal ammonia
(C) Direct caustic action of un-dissociated lactic acid
(D) Mechanical injury from abrasive dry forage roughage

2007
181. Mycotic rumenitis often follows chemical rumenitis. The pathognomonic lesion characterizing fungal invasion of the ruminal wall is:
(A) Hemorrhagic infarction and submucosal thrombosis
(B) Diffuse mucosal hyperplasia with epithelial scaling
(C) Caseous granulomas located within the deep muscle layer
(D) Suppurative microabscesses lining the superficial mucosa

2001
182. The fungal species most commonly responsible for mycotic rumenitis following acidosis belong to the genera:
(A) Microsporum and Trichophyton
(B) Candida and Malassezia
(C) Cryptococcus and Histoplasma
(D) Mucor, Rhizopus, and Aspergillus

2009
183. Sudden death with massive epistaxis in a steer with a history of grain overload is classically linked to:
(A) Direct fungal erosion of the common carotid artery in the neck
(B) Pulmonary artery aneurysm rupture secondary to caval thrombosis
(C) Traumatic hepatic rupture with massive intra-abdominal bleeding
(D) Systemic coagulopathy arising from dietary vitamin K deficiency

2008
184. Which step is INCORRECTLY described in the pathogenesis of the rumenitis-liver abscess complex?
(A) Lactic acid induces necrosis of the ruminal epithelium.
(B) Rumen bacteria establish colonies in the damaged epithelium.
(C) Bacteria travel via the hepatic vein to colonize the liver.
(D) F. necrophorum elaborates a leukotoxin that protects it from phagocytosis in the liver.

2001
185. Which organism is LEAST likely to be a primary pathogen in the rumenitis-liver abscess complex?
(A) Fusobacterium necrophorum
(B) Trueperella pyogenes
(C) Bacteroides species
(D) Mycobacterium bovis

NODIA

2016
186. Primary ruminal tympany (frothy bloat) is most commonly triggered by the ingestion of:
(A) Lush legumes high in soluble leaf proteins
(B) High-fiber, poor-quality stemmy roughage
(C) Excessive amounts of grain concentrate feed
(D) Toxic pasture plants containing high cyanide

2013
187. The classic gross post-mortem lesion used to diagnose fatal bloat is:
(A) Massive hemorrhagic lesions within the mucosa of the abamasal wall
(B) An esophageal "bloat line" with cranial congestion and caudal pallor
(C) Spontaneous post-mortem rupture of the splenic capsule and parenchyme
(D) Diffuse fibrinopurulent peritonitis throughout the abdominal cavity

of secretory epithelia, such as columnar or transitional cells. Without Vitamin A, these cells are replaced by stratified, keratinized squamous epithelium, which compromises local defense mechanisms and salivary or mucous secretion.

38. (A) Both A and R are true and R is the correct explanation of A.
Vitamin A deficiency leads to nyctalopia because retinal, specifically the 11-cis-retinal isomer, is the crucial light-absorbing chromophore that combines with opsin to form rhodopsin. Rhodopsin is the highly sensitive photopigment situated within the rod cells of the retina, responsible for low-light vision. Absent sufficient retinal, rhodopsin synthesis fails, preventing proper adaptation to darkness.

39. (B) Vitamin A
Maternal Vitamin A deficiency during gestation disrupts critical developmental pathways in the fetus, particularly the signaling networks regulating organogenesis. Retinoic acid is essential for the normal morphogenesis of the ocular structures. A severe deficiency of this vitamin during early pregnancy frequently results in congenital ophthalmic defects in calves, most classically microphthalmia.

40. (C) Keratinized epithelial debris
In animals with Vitamin A deficiency, the normal bi-layered columnar epithelium lining the parotid salivary duct undergoes squamous metaplasia. This newly formed stratified squamous epithelium undergoes abnormal keratinization, sloughing large amounts of keratinized debris into the narrow lumen. This accumulating mass of dead cells and keratin occludes the duct, leading to salivary stasis and cystic dilation.

41. (D) Impaired sphenoid bone remodeling
Vitamin A deficiency in growing animals impairs the coordinated osteoclastic and osteoblastic remodeling of the skull bones. Specifically, the sphenoid bone surrounding the optic canal fails to resorb properly, causing the canal to remain narrow while the nerve continues to grow. This osseous constriction compresses the optic nerve, leading to ischemic necrosis and blindness.

42. (A) Keratinized and lacks cilia
Under normal conditions, the respiratory tract is lined by pseudostratified ciliated columnar epithelium. Vitamin A deficiency alters epithelial differentiation, causing this specialized lining to undergo squamous metaplasia. The resulting metaplastic epithelium is stratified, heavily keratinized, and completely lacks cilia. This structural change severely compromises the mucociliary escalator, leaving the animal highly vulnerable to respiratory infections.

43. (B) Osteomalacia
Vitamin A deficiency is classically associated with squamous metaplasia, xerophthalmia, and nyctalopia, which reflect the essential role of retinoids in epithelial differentiation and visual pigment synthesis. In contrast, osteomalacia is a bone disorder characterized by the accumulation of unmineralized osteoid, which is directly caused by a deficiency of Vitamin D or phosphorus, not Vitamin A.

44. (B) Beta-carotene : Active visual pigment
Beta-carotene is a carotenoid precursor, or provitamin, that must be enzymatically cleaved in the intestinal mucosa to yield active Vitamin A. It does not function directly as the active visual pigment. Retinal, specifically 11-cis-retinal, is the actual visual chromophore, while retinoic acid regulates transcription for epithelial differentiation, and retinol serves as the primary transport form.

45. (A) P-4, Q-1, R-2, S-3
Squamous metaplasia affects the mucosal linings of respiratory and glandular ducts. Nyctalopia stems from rhodopsin depletion in the photoreceptive retina. Optic nerve constriction occurs due to failed remodeling of the sphenoid bone foramen in growing animals. Microphthalmia is a congenital defect resulting from disrupted retinoic acid signaling during fetal ocular development in utero.

46. (C) Rickets
In young, growing animals with active epiphyseal plates, Vitamin D deficiency leads to rickets, a disease defined by defective mineralization of bone osteoid and cartilaginous matrices. Without adequate Vitamin D, intestinal absorption of calcium and phosphorus is compromised. This systemic mineral deficiency prevents normal provisional calcification at the physis, leading to weak, deformed bones.

Click Here to Buy book on Amazon

phosphorus drain.
(B) High dietary calcium limits intestinal phosphorus absorption.
(C) The primary defect is a lack of renal erythropoietin production.
(D) Spherocytes are easily destroyed within the microvasculature.

2005
201. Which process does NOT occur as a result of erythrocyte ATP depletion in this disease?
(A) Intracellular accumulation of sodium and water
(B) Loss of structural membrane deformability
(C) Eventual rupture of the plasma membrane
(D) Immune-complex deposition on the membrane

2008
202. In a fatal case of post-parturient haemoglobinuria, the kidneys grossly exhibit a dark, red-to-black discoloration often referred to as:
(A) "Gunmetal kidneys"
(B) "White spotted kidneys"
(C) "Pulpy kidneys"
(D) "Flea-bitten kidneys"

2018
203. The severe centrilobular hepatic necrosis observed on necropsy of these cases is a direct result of:
(A) Viral hepatitis
(B) Severe anemic hypoxia
(C) Copper toxicity
(D) Direct phosphorus toxicity to hepatocytes

2007
204. The severe icterus (jaundice) seen in post-parturient haemoglobinuria is mechanistically classified as:
(A) Post-hepatic (obstructive)
(B) Pre-hepatic (hemolytic)
(C) Hepatic (toxic)
(D) Neonatal isoerythrolysis

2015
205. Histological examination of the kidney in hemoglobinuric nephrosis reveals:
(A) Brightly eosinophilic hemoglobin casts in the tubules
(B) Massive extracellular amyloid deposition in the glomeruli
(C) Suppurative interstitial nephritis within the renal cortex
(D) Marked proliferation of the mesangial cells in glomerular loops

2008
206. Over time, animals recovering from severe intravascular hemolysis will show massive accumulation of which pigment in the spleen and liver?
(A) Lipofuscin
(B) Melanin
(C) Hemosiderin
(D) Ceroid

2025
207. Which characteristic reliably distinguishes hemoglobinuria from hematuria upon physical examination of the urine?
(A) Hemoglobinuric urine will clear completely upon centrifugation, leaving a red pellet.
(B) Hematuria never causes the urine to appear red.
(C) Hematuria is always accompanied by pyuria.
(D) Hemoglobinuric urine remains uniformly red/brown after centrifugation.

2016
208. Identify the INCORRECT pairing between a lesion of post-parturient haemoglobinuria and its direct cause.
(A) Hemoglobinuric nephrosis : Deposition of free hemoglobin in tubules
(B) Icterus : Overwhelmed hepatic bilirubin conjugation capacity
(C) Splenomegaly : Primary lymphoid neoplasia
(D) Centrilobular necrosis : Acute severe anemia

2017
209. In severe hemoglobinuric nephrosis, routine urinalysis will most likely reveal:
(A) High specific gravity with massive glycosuria in the presence of ketones
(B) Isosthenuria and positive occult blood tests without intact erythrocytes
(C) Low specific gravity with extreme ketonuria and high concentrations of glucose
(D) Normal specific gravity with massive numbers of intact cellular erythrocytes

NODIA

2024
210. Ultimately, the systemic collapse and death in terminal post-parturient haemoglobinuria are driven by the culminating effects of:
(A) Hypocalcemic tetany and cardiac arrest
(B) Massive gastrointestinal hemorrhage
(C) Encephalomalacia and brain herniation
(D) Severe anemic anoxia and acute renal failure

****

2015

167. Why do adult ruminants develop hypomagnesemia so rapidly when dietary intake drops?
(A) Their kidneys excrete magnesium actively regardless of blood levels.
(B) They lack a mobilizable hormonal reserve of magnesium in bone.
(C) The rumen microbes consume all available magnesium.
(D) Magnesium is entirely secreted into milk within hours.

2002

168. Which statement regarding magnesium metabolism is INCORRECT?
(A) Absorption occurs primarily across the ruminal epithelium in adult ruminants.
(B) The transport of magnesium across the rumen wall is active and sodium-linked.
(C) Parathyroid hormone rapidly mobilizes magnesium from adult bones.
(D) High ruminal ammonia levels can severely interfere with magnesium absorption.

2022

169. Which condition is LEAST likely to precipitate hypomagnesemic tetany?
(A) Acute physiological transport stress
(B) Sudden exposure to cold, wet weather
(C) Grazing lush, rapidly growing spring grass
(D) High intake of mature, dry hay

2003

170. Acute ruminal acidosis (Grain overload) is initiated by the rapid fermentation of highly digestible carbohydrates, leading to a massive proliferation of which bacteria?
(A) Streptococcus bovis
(B) Fibrobacter succinogenes
(C) Ruminococcus albus
(D) Fusobacterium necrophorum

2006

171. A profound shift in ruminal microflora during grain overload is characterized by:
(A) A decrease in Gram-positive species with a concurrent increase in Gram-negative species
(B) A shift from Gram-negative flora to lactic-acid-producing Gram-positive bacteria
(C) A rapid proliferation in the population of cellulolytic Gram-negative bacteria
(D) Complete and irreversible sterilization of all viable ruminal microflora

2011

172. Systemic acidosis in grain overload is particularly severe and prolonged due to the systemic absorption of which specific isomer of lactic acid, which ruminants metabolize slowly?
(A) L-lactate
(B) D-lactate
(C) DL-lactate
(D) Pyruvic acid

2004

173. The profound dehydration observed in acute ruminal acidosis is primarily driven by:
(A) Elevated ruminal fluid osmolarity, drawing water from circulation into the rumen
(B) Excessive and prolonged sweating caused by elevated systemic core body temperature
(C) Massive renal excretion of metabolic water attempting to clear systemic organic acids
(D) Severe internal hemorrhage from gastric ulceration within the abamasum

2015

174. Which event happens relatively early in the pathogenesis of acute ruminal acidosis as the pH drops below 5.5?
(A) Proliferation of methanogenic archaea
(B) Hypertrophy of ruminal papillae
(C) Death and lysis of ruminal protozoa
(D) Regeneration of ruminal epithelium

2015

175. The systemic acid-base disturbance in severe grain overload is best characterized biochemically as:
(A) Normal anion gap metabolic acidosis
(B) Respiratory acidosis
(C) Metabolic alkalosis
(D) High anion gap metabolic acidosis

2006

176. Which pathophysiological change is INCORRECTLY attributed to grain overload?
(A) Hemoconcentration and increased hematocrit
(B) Ruminal stasis
(C) Increased ruminal pH
(D) Decreased systemic blood pressure (shock)

2005

177. Which clinical manifestation is NOT a direct consequence of acute ruminal acidosis?
(A) Acute renal anuria or oliguria
(B) Intravascular hemolysis
(C) Bilateral scleral congestion
(D) Acute systemic laminitis

Downloaded from www.pdf.tube

9. (D) Neoplasia
Non-carcinogenic nutritional deficiencies elicit physiological adaptations such as cellular atrophy, autophagy, or metaplasia, helping cells survive metabolic stress. In contrast, neoplasia is an uncontrolled, autonomous, and irreversible proliferation of abnormal cells triggered by genetic mutations rather than simple nutritional deprivation. Therefore, neoplasia is not a direct or expected physiological adaptation to a clean dietary deficit.

10. (A) Serous atrophy of fat
During chronic starvation, the body sequentiality mobilizes adipose tissue depots to provide energy, depleting vital cardiac fat reserves last. At necropsy, the coronary groove displays serous atrophy of fat, where depleted lipid deposits are replaced by a gelatinous, watery, translucent mass. This lesion represents the physical collapse of adipocytes and fluid accumulation in empty spaces.

11. (B) I and III
Cachexia is a complex wasting syndrome driven by pro-inflammatory cytokines such as tumor necrosis factor-alpha. Unlike simple starvation, which conserves energy, cachexia accelerates the basal metabolic rate, causing profound and concurrent loss of both skeletal muscle and adipose tissue. Skeletal muscle mass is actively degraded and is never preserved in this cytokine-driven metabolic state.

12. (B) Mucoid extracellular fluid
Severe protein-energy malnutrition forces the complete mobilization of adipose tissue within the bone marrow, which represents one of the final lipid stores. As the adipocytes lose their fat content and shrink, the remaining empty interstitial spaces are filled with mucoid extracellular fluid rich in mucopolysaccharides. This produces a characteristic gelatinous, translucent, and watery bone marrow.

13. (A) Marked lymphoid atrophy
Severe protein-energy malnutrition deprives the body of essential building blocks required for rapid cellular division and protein synthesis. Consequently, lymphoid organs, such as the thymus, spleen, and lymph nodes, undergo marked lymphoid atrophy. This structural depletion of lymphocytes impairs cell-mediated and humoral immunity, rendering the malnourished animal highly susceptible to opportunistic systemic infections.

14. (C) Subcutaneous fat depot
In the pathogenesis of progressive starvation, the body mobilizes fat depots in a highly organized, sequential manner to meet energy needs. The subcutaneous adipose tissue is mobilized first, followed later by internal visceral depots like omental and perirenal fat. The bone marrow and epicardial fat depots are exceptionally preserved, undergoing depletion only during terminal starvation.

15. (D) Skeletal muscle proteins
When glycogen reserves and primary adipose tissue depots are exhausted during progressive starvation, the body must utilize alternative energy substrates. To preserve vital brain and organ functions, the endocrine system promotes the extensive catabolism of skeletal muscle proteins. This systemic breakdown releases amino acids for hepatic gluconeogenesis, producing the profound muscle wasting characteristic of terminal starvation.

16. (C) Kwashiorkor-like conditions feature selective, severe protein deficiency.
In veterinary pathology, kwashiorkor-like syndromes are characterized by selective, severe protein deficiency occurring in animals with relatively adequate caloric intake. This is distinguished from marasmus-like conditions, which are defined by balanced starvation involving severe deficiencies of both protein and total dietary energy. The severe hypoproteinemia in kwashiorkor leads to edema, hepatomegaly, and abdominal distension.

17. (B) Adipocytes are hyperplastic with large lipid droplets.
During serous atrophy of fat, adipocytes undergo severe regression, shrinking into small, stellate, or spindle-shaped cells as they deplete their internal lipid reserves. They are never hyperplastic and do not contain large lipid droplets. The surrounding interstitial space becomes filled with a watery, pale eosinophilic mucoid fluid, making the underlying capillary network appear prominent.

NODIA

18. (D) II, III, I
The mobilization of fat depots during starvation follows a highly predictable, ordered sequence to maintain systemic energy homeostasis.

ANSWERS

1. (B) ATP depletion causing failure of the sodium-potassium ATPase pump
Severe nutritional deprivation limits the cellular substrates required for oxidative phosphorylation, resulting in acute depletion of adenosine triphosphate. Without adequate energy, the sodium-potassium adenosine triphosphatase pump fails, preventing the active extrusion of sodium ions. Consequently, intracellular sodium accumulates, drawing water into the cytosol along the osmotic gradient, leading to cell swelling.

2. (D) Atrophy
Atrophy is a reversible adaptive response characterized by a decrease in cellular size and number, leading to reduced organ volume. When metabolic resources are scarce, cells downregulate energy demand by digesting their own structural proteins and organelles. This controlled reduction balances the metabolic needs of the remaining cell structures to maximize survival.

3. (A) Intracellular calcium levels strictly remain below normal limits.
Irreversible cellular injury is fundamentally associated with a profound influx of calcium into the cytosol, raising intracellular calcium concentrations far above normal physiological limits. This abnormal elevation activates destructive calcium-dependent enzymes, including phospholipases and proteases, which permanently damage vital membranes and structural proteins, eventually committing the cell to necrotic or apoptotic death.

4. (C) Apoproteins
Severe or prolonged dietary protein deficiency directly limits the availability of amino acids necessary for translation, impairing the synthesis of apoproteins in hepatocytes. Apoproteins are required to bind with intracellular lipids to assemble very-low-density lipoproteins. In their absence, lipids cannot be exported from the hepatic cells, leading to severe cellular accumulation of triglycerides.

5. (A) Both A and R are true and R is the correct explanation of A.
Chronic starvation deprives cells of exogenous nutrients, forcing parenchymal cells to activate autophagocytosis as a vital survival mechanism. This physiological process sequesters non-vital intracellular components, such as excess organelles, within double-membraned vesicles that fuse with lysosomes. The subsequent enzymatic degradation releases free amino acids and metabolic substrates to sustain life-sustaining cellular functions.

6. (D) Mitochondria
Severe nutritional anemia causes a profound reduction in oxygen-carrying capacity, inducing secondary hypoxia that heavily impacts aerobic cellular metabolism. Mitochondria, being the principal sites of oxygen utilization for the electron transport chain, are highly sensitive to oxygen deprivation. Hypoxia immediately arrests mitochondrial oxidative phosphorylation, causing structural swelling and functional collapse of these powerhouses.

7. (B) Metaplasia
Metaplasia is a controlled, reversible adaptive change in which one mature, differentiated cell type is replaced by another differentiated cell type. This alteration typically occurs in response to chronic environmental or nutritional stress, enabling the tissue to withstand harsh conditions. The newly formed cell type is more resilient, though it frequently lacks the original specialized functions.

8. (C) Fatty acid oxidation and lipoprotein export
Intracellular accumulation of lipid vacuoles, or steatosis, primarily occurs in organs like the liver when metabolic pathways are disrupted. This pathologic accumulation indicates a severe imbalance where hepatocytes fail to execute mitochondrial fatty acid beta-oxidation or synthesize sufficient apoproteins. Consequently, lipids cannot be oxidized or packaged into lipoproteins for export, causing accumulation within hepatocytes.

Click Here to Buy book on Flipkart

28. (B) Piglets
Neonatal piglets are exceptionally vulnerable to severe, life-threatening hypoglycemia because they are born with minimal adipose tissue and extremely limited hepatic glycogen reserves. They depend almost entirely on immediate, frequent colostrum and milk intake to maintain normoglycemia. If feeding is delayed or interrupted, their small glycogen reserves are exhausted within hours, inducing acute hypoglycemia.

29. (A) Gluconeogenesis
During the first few days of life, piglets have an immature hepatic system that cannot efficiently execute gluconeogenesis. They cannot synthesize glucose from non-carbohydrate precursors like amino acids or lactate to compensate for fasting. Consequently, if colostrum intake is disrupted, they cannot maintain blood glucose levels once their limited glycogen stores are exhausted.

30. (C) III only
Neonatal piglets have an immature hepatic system with highly inefficient gluconeogenesis during their first days. Consequently, blood glucose levels cannot be maintained and fall rapidly during fasting. Maternal agalactia directly triggers this crisis, while hypothermia accelerates energy depletion by forcing shivering. Clinical signs include shivering, weak locomotion, convulsions, and eventually fatal coma.

31. (D) Encephalopathy and coma
Untreated neonatal hypoglycemia in piglets, historically termed baby pig disease, leads directly to neuroglycopenia. Because the neonatal brain relies almost exclusively on circulating glucose as its primary energy source, prolonged deficits compromise neuronal function and survival. This metabolic failure manifests clinically as progressive encephalopathy, starting with weakness and convulsions, and culminating in fatal coma.

32. (C) Glycogen depletion
At necropsy, the most significant and consistent finding in a neonate that died of acute hypoglycemia is complete glycogen depletion in the liver. Because the animal was unable to obtain or absorb sufficient nutrients, its hepatocytes mobilized all stored glycogen in a failed physiological attempt to maintain systemic normoglycemia, leaving the liver structurally depleted of glycogen.

33. (B) Glucose
The neonatal brain is highly active and metabolically demanding, relying almost exclusively on circulating blood glucose to fuel its cellular processes. Unlike adult brains, neonates cannot efficiently utilize alternative fuels such as ketone bodies. Consequently, when maternal agalactia deprives the neonate of milk, blood glucose drops, rapidly starving the brain and causing terminal encephalopathy.

34. (D) Shivering rapidly depletes glucose.
Hypothermia places extreme physiological stress on neonates, who must maintain core body temperature. To generate heat, the body initiates shivering, an involuntary muscular activity that is highly energy-demanding. This rapid increase in muscle contraction quickly depletes the limited glycogen and glucose reserves of the animal, thereby accelerating the onset and severity of life-threatening hypoglycemia.

35. (A) Cortical neuronal degeneration
Prolonged hypoglycemic coma deprives cerebral neurons of the glucose essential for maintaining membrane potentials and cellular integrity. Histopathological examination of the brain under these conditions reveals acute, diffuse cortical neuronal degeneration. Affected neurons exhibit classic changes of ischemic-type necrosis, including cellular shrinkage, marked cytoplasmic hypereosinophilia, and nuclear pyknosis or karyorrhexis.

36. (C) Fasting → Depletion of hepatic glycogen → Failure of gluconeogenesis → Neuroglycopenia
The fatal pathogenesis begins with fasting due to starvation or maternal agalactia. The piglet first mobilizes its limited carbohydrate reserves, causing rapid depletion of hepatic glycogen. Because the immature liver cannot perform gluconeogenesis efficiently, blood glucose levels plummet. This systemic deficit leads directly to neuroglycopenia, starving the brain and causing terminal encephalopathy and coma.

37. (A) Epithelial squamous metaplasia
The hallmark histological lesion of Vitamin A deficiency is epithelial squamous metaplasia across various mucosal surfaces. Retinoids are required to maintain the normal differentiation

Downloaded from www.pdf.tube

2008
188. The immediate cause of death in severe ruminal tympany is:
(A) Acute traumatic rupture of the dorsal ruminal sac
(B) Asphyxiation and collapse from diaphragmatic compression
(C) Systemic toxemia from absorption of noxious ruminal gases
(D) Secondary severe systemic lactic metabolic acidosis

2011
189. Secondary ruminal tympany (free-gas bloat) fundamentally results from:
(A) Failure of the eructation mechanism
(B) Overproduction of foam in the rumen
(C) Excessive consumption of water
(D) Complete absence of ruminal fermentation

2005
190. A common etiology of chronic secondary bloat is:
(A) Primary dietary lack of structural fiber
(B) Overeating high-starch grain concentrates
(C) Vagal nerve injury or mediastinal tissue compression
(D) High dietary intake of immature legumes

2009
191. In frothy bloat, the stable foam that traps fermentation gases is stabilized by:
(A) Persistently elevated ruminal pH levels
(B) Soluble cell-wall bacterial endotoxins
(C) Salivary bicarbonate acting as a buffer
(D) Plant proteins, saponins, and pectins

2010
192. Which gross post-mortem finding is INCORRECTLY associated with death by ruminal tympany?
(A) The lungs are severely compressed, consolidated, and atelectatic.
(B) The cranial cervical and thoracic organs are severely congested.
(C) The hindquarters are congested while the forequarters are pale.
(D) The ruminal mucosa peels away easily from the muscular wall.

2007
193. Which of the following conditions is LEAST likely to cause secondary ruminal tympany?
(A) Esophageal obstruction (choke)
(B) Ingestion of high-legume pasture
(C) Tetanus
(D) Reticulitis causing reticular adhesions

2011
194. Post-parturient haemoglobinuria in high-yielding dairy cows is fundamentally driven by a severe acute deficiency of:
(A) Dietary iron
(B) Calcium
(C) Magnesium
(D) Phosphorus

2025
195. The hallmark pathological event defining post-parturient haemoglobinuria is:
(A) Massive acute intravascular hemolysis
(B) Immune-mediated extravascular hemolysis
(C) Hemorrhage into the urinary bladder
(D) Severe bone marrow aplasia

2001
196. Severe hypophosphatemia leads to erythrocyte lysis by causing a critical depletion of:
(A) Protective cellular glutathione peroxidase
(B) Active erythrocytic methemoglobin reductase
(C) Intracellular ATP and Na^+/K^+ pump function
(D) Structural erythrocyte membrane cholesterol

2006
197. The depletion of erythrocyte ATP causes the cell to lose its biconcave shape and form a:
(A) Fragile spherocyte
(B) Fragmented schistocyte
(C) Spiculated acanthocyte
(D) Leptocytic target cell

2023
198. Post-parturient haemoglobinuria typically occurs during which phase of the cow's production cycle?
(A) Immediately pre-partum prior to calving
(B) Within twenty-four hours of calving
(C) During the non-lactating dry period
(D) Two to four weeks following calving

NODIA

2017
199. Which pathological feature is INCORRECTLY associated with post-parturient haemoglobinuria?
(A) Markedly decreased packed cell volume (PCV)
(B) Formation of Heinz bodies on erythrocytes
(C) Hemoglobinemia
(D) Hemoglobinuria

2010
200. Which statement INCORRECTLY outlines the pathogenesis of post-parturient haemoglobinuria?
(A) High milk yield acts as a massive maternal

47. (D) Defective mineralization of osteoid
The primary pathological defect in both rickets and osteomalacia is the failure to mineralize newly formed bone matrix (osteoid). Osteoblasts continue to synthesize and deposit the organic collagenous matrix, but the lack of sufficient calcium or phosphorus prevents hydroxyapatite crystal deposition. This leads to an abnormal accumulation of soft, unmineralized osteoid seams on bone surfaces.

48. (C) I, II, III, and V
Vitamin D promotes the intestinal absorption of minerals, and its deficiency in young animals impairs provisional calcification during endochondral ossification, causing rickets with widened, irregular epiphyseal plates. In adults, it manifests as osteomalacia, with unmineralized osteoid accumulating on trabecular surfaces. The active form of the vitamin is 1,25-dihydroxycholecalciferol, not 25-hydroxycholecalciferol.

49. (B) Enlargement of costochondral junctions due to accumulation of unmineralized osteoid and cartilage
The rachitic rosary is a classic gross skeletal lesion of rickets, visible along the rib cage. It is characterized by prominent, nodular enlargements of the costochondral junctions. This swelling is directly caused by the accumulation of excess, unmineralized osteoid and persistent, hypertrophic cartilage that fails to undergo normal provisional calcification and subsequent osteoclastic resorption.

50. (D) Absent provisional calcification of cartilage
Microscopically, the epiphyseal plate of an animal with rickets displays a failure of provisional calcification in the cartilage matrix. Because the matrix does not mineralize, blood vessels cannot invade the zone of hypertrophy, preventing the normal replacement of cartilage by bone. This leads to an abnormal, irregular accumulation of chondrocytes and unmineralized osteoid.

51. (A) 1,25-dihydroxycholecalciferol
The active hormone regulating calcium homeostatic pathways is 1,25-dihydroxycholecalciferol, synthesized in the kidneys from its precursor, 25-hydroxycholecalciferol, via the enzyme 1-alpha-hydroxylase. This potent metabolite binds to nuclear Vitamin D receptors in enterocytes, upregulating the transcription of calcium-binding proteins, such as calbindin, to facilitate active intestinal absorption of calcium and phosphorus.

52. (C) Lactating or pregnant adult animals
Osteomalacia occurs in mature, adult animals after the closure of the epiphyseal growth plates. It is most frequently observed in lactating or pregnant females due to the high physiological demand for calcium and phosphorus required for milk production and fetal skeletal development. These intense metabolic drains exhaust mineral reserves, leading to defective remodeling of bone.

53. (A) The physeal growth plates are markedly thickened.
Because osteomalacia develops only in mature, adult animals after the growth plates have already closed, there are no physeal growth plates to become thickened. This lack of active physes distinguishes osteomalacia from rickets. Microscopic evaluation instead reveals the accumulation of wide, unmineralized osteoid seams on the surfaces of mature cortical and trabecular bone.

54. (A) Both A and R are true and R is the correct explanation of A.
In rickets, endochondral ossification at the physis is severely disrupted because the provisional matrix of cartilage fails to mineralize. Normal mineralization is the essential biochemical signal that triggers vascular invasion. Without this calcification, capillaries cannot penetrate the hypertrophic zone, preventing osteoblasts from depositing bone on cartilage templates and causing an irregular accumulation of cartilage.

55. (B) Striated muscle tissue
White Muscle Disease is a nutritional degenerative myopathy that selectively damages striated muscle tissue, which includes both skeletal and cardiac muscles. These tissues undergo extensive lipid peroxidation of their cell membranes due to a deficiency in antioxidant protection. This damage leads to segmentary hyaline necrosis and mineralization, giving affected muscles a pale, chalky, white appearance.

56. (C) Swine
Mulberry Heart Disease is a distinct pathological condition that occurs almost exclusively in swine. It is caused by a nutritional deficiency of Vitamin E or Selenium, which leads to widespread

Click Here to Buy book on Amazon

95. (D) Cerebral white matter cavitation
In severe congenital swayback, necropsy of affected lambs reveals cerebral white matter cavitation as a characteristic gross lesion. This lesion represents the physical collapse of myelin-deficient cerebral tissue, forming bilateral, fluid-filled cavities in the subcortical white matter. This profound structural defect is a direct consequence of severe prenatal copper deficiency compromising early oligodendroglial development.

96. (C) Massive neutrophil infiltration
The spinal cord of lambs with enzootic ataxia displays degenerative changes, including motor neuron degeneration in the ventral horns, Wallerian-type degeneration, and demyelination of descending tracts. However, massive neutrophil infiltration is not observed, as enzootic ataxia is a non-inflammatory, metabolic, and myelinogenic deficiency disease rather than an acute infectious or purulent process.

97. (B) Molybdenum and Sulfur
Secondary copper deficiency in ruminants is commonly induced by excessive dietary levels of molybdenum and sulfur. In the rumen, these minerals react to form thiomolybdates, which bind copper ions to create insoluble copper thiomolybdate complexes. This biochemical binding prevents copper absorption in the gut, inducing a functional systemic deficiency despite adequate dietary copper concentrations.

98. (D) Glutathione peroxidase
Ceruloplasmin, cytochrome c oxidase, and lysyl oxidase are all classic copper-dependent metalloenzymes whose activities are directly impaired during copper deficiency. In contrast, glutathione peroxidase is a selenium-dependent metalloenzyme that utilizes selenium as an essential cofactor to destroy intracellular peroxides. It does not require copper for its synthesis or its enzymatic function.

99. (A) Low iron in sow's milk and no soil access
The primary etiology of piglet anemia is the exceptionally rapid growth rate of neonatal pigs combined with extremely low iron levels in sow's milk. Piglets kept on concrete floors without access to soil cannot obtain exogenous iron. Without iron supplementation, their small hepatic iron reserves are exhausted within days, causing a severe microcytic hypochromic anemia.

100. (C) I and III
Iron deficiency anemia in piglets is a classic microcytic, hypochromic anemia caused by impaired hemoglobin synthesis. It is characterized by oligocythemia and reduced packed cell volume, not polycythemia. Affected piglets show pale mucous membranes, lethargy, poor growth, and a characteristic spasmodic breathing pattern known as "thumps" due to chronic tissue hypoxia.

101. (A) Cobalt
Pine is a wasting disease in sheep and cattle directly caused by cobalt deficiency. Cobalt is an essential substrate required by ruminal microorganisms to synthesize Vitamin B12 (cyanocobalamin). In cobalt-deficient pastures, microbes fail to produce this vitamin, leading to a systemic Vitamin B12 deficiency that impairs energy metabolism and produces progressive emaciation.

102. (D) Cyanocobalamin
Cobalt is an essential trace element that serves as the central metal atom in the corrin ring of cyanocobalamin (Vitamin B12). Ruminant animals rely entirely on the synthetic activity of their ruminal microflora to incorporate cobalt into this vitamin. Without sufficient cobalt, microflora cannot produce Vitamin B12, causing deficiency signs in the host.

NODIA

103. (B) Small size and pale central pallor
In a peripheral blood smear of an iron-deficient piglet, erythrocytes appear microcytic and hypochromic, presenting as small cells with an exaggerated, pale central pallor. This morphology reflects the lack of intracellular iron, which prevents the proper synthesis and concentration of hemoglobin within developing red blood cells, resulting in thin, pale-staining erythrocytes.

104. (C) Cobalt deficiency
White liver disease in sheep is a pathological condition characterized by severe hepatic lipidosis resulting from chronic cobalt deficiency. Without cobalt, ruminal microbes cannot synthesize Vitamin B12, which is a cofactor for propionate metabolism. This metabolic blockade disrupts gluconeogenesis and fat transport, causing triglycerides to accumulate within hepatocytes, producing a pale, fatty liver.

66. (D) Riboflavin
Curled-toe paralysis is a highly characteristic pathognomonic clinical sign observed in growing chicks deficient in riboflavin (Vitamin B2). Riboflavin is crucial for the synthesis of flavin adenine dinucleotide and flavin mononucleotide. Its deficiency leads to myelin sheath degeneration and Schwann cell swelling in peripheral nerves, particularly the sciatic nerve, causing the characteristic inward curling of the toes.

67. (A) Sciatic nerve Schwann cell hyperplasia
The neurological lesion responsible for curled-toe paralysis involves demyelination and Schwann cell hypertrophy and hyperplasia in peripheral nerves, particularly the sciatic and brachial nerves. Under microscopic examination, these nerves display swelling, axis cylinder degeneration, and a marked proliferative response of the myelin-producing Schwann cells, which directly disrupts motor impulses and leads to paralysis.

68. (C) Thiamine deficiency
In poultry, thiamine deficiency classically manifests as systemic polyneuritis. This nutritional deficiency disrupts carbohydrate metabolism in nervous tissues, which are highly dependent on glucose for energy. The resulting metabolic failure leads to progressive demyelination and degeneration of peripheral nerves. Affected birds exhibit retraction of the head (opisthotonos), weakness, and ascending paralysis of the limbs.

69. (A) Necrotic cerebral cortex fluorescence
A unique diagnostic feature of acute polioencephalomalacia in ruminants is the autofluorescence of the necrotic cerebral cortex when examined under ultraviolet light. The degenerating cortical tissues accumulate lipofuscin and lipid breakdown products within macrophages and necrotic cells. These lipopigments fluoresce intensely under ultraviolet illumination, providing a rapid post-mortem method to confirm cerebrocortical necrosis.

70. (D) Cerebellar peduncle demyelination
Polioencephalomalacia is defined as necrosis of the cerebral cortex gray matter, characterized microscopically by spongiosis, neuronal necrosis with pyknotic nuclei, and gitter cell accumulation. It selectively spares the white matter tracts and hindbrain structures. Therefore, demyelination of the cerebellar peduncle is not a feature of this disease, representing a different neurological distribution.

71. (B) Enzootic ataxia
While curled-toe paralysis, black tongue, and polioencephalomalacia are classic manifestations of riboflavin, niacin, and thiamine deficiencies, respectively, enzootic ataxia is not related to Vitamin B. Enzootic ataxia is a copper deficiency syndrome in lambs that leads to demyelination of the spinal cord due to the failure of copper-dependent enzymes like cytochrome c oxidase.

72. (C) P-2, Q-1, R-4, S-3
Thiamine deficiency causes polioencephalomalacia in ruminants. Riboflavin deficiency results in curled-toe paralysis in chicks. Niacin deficiency causes black tongue in dogs, which is the canine equivalent of pellagra. Cyanocobalamin deficiency occurs secondary to Cobalt deficiency in ruminants, as ruminal microbes require Cobalt to synthesize Vitamin B12, causing severe systemic wasting.

73. (D) II, VII, IX, X
Vitamin K serves as an indispensable cofactor for the post-translational modification of specific clotting factors in the liver, namely Factors II (prothrombin), VII, IX, and X. These proteins require Vitamin K-dependent gamma-carboxylation of their glutamic acid residues, which allows them to bind calcium ions and attach to phospholipid membranes, initiating effective blood coagulation.

74. (A) I, II, and III
Dicoumarol is a potent Vitamin K antagonist that inhibits the enzyme vitamin K epoxide reductase, preventing the recycling of active Vitamin K. This biochemically arrests the gamma-carboxylation of Factors II, VII, IX, and X, leading to severe deficiency of active clotting factors. Consequently, this causes a profound hemorrhagic diathesis, not intravascular thrombosis.

75. (B) Moldy sweet clover
Sweet clover poisoning in cattle is caused by the ingestion of moldy sweet clover hay. During spoilage, natural coumarins in the plant are converted by fungi into dicoumarol, a highly active Vitamin K antagonist. Ingesting this compound inhibits vitamin K epoxide reductase, leading to

Downloaded from www.pdf.tube

microangiopathy, vascular thrombosis, and myocardial necrosis. Grossly, the epicardium and myocardium display extensive, dark red hemorrhages, giving the heart an appearance resembling a mulberry.

57. (D) P-3, Q-4, R-2, S-1
White Muscle Disease causes skeletal myonecrosis in calves and lambs. Exudative Diathesis causes severe subcutaneous edema in poultry due to capillary damage. Mulberry Heart Disease produces microangiopathy and myocardial hemorrhage in swine. Encephalomalacia causes cerebellar necrosis, known as crazy chick disease, in poultry. Each condition represents tissue-specific oxidative damage resulting from a lack of cellular antioxidants.

58. (A) Free radical damage to capillary endothelium
Exudative diathesis in chicks is a manifestation of Vitamin E and Selenium deficiency where cellular membranes are left unprotected from oxidative stress. Free radicals attack the lipid bilayers of the capillary endothelium, leading to widespread microvascular damage and increased permeability. This allows plasma to leak extensively into the subcutaneous tissues, resulting in massive, gelatinous edema.

59. (B) Glutathione peroxidase
Selenium is an essential trace mineral that serves as a critical cofactor for glutathione peroxidase, a major cytosolic antioxidant enzyme. This enzyme catalyzes the reduction of harmful hydrogen peroxide and lipid hydroperoxides into water and alcohols, utilizing reduced glutathione. By destroying these reactive species, it prevents free radical damage to cell membrane lipids.

60. (B) Synergistic antioxidants
Vitamin E and Selenium act as synergistic antioxidants that collaborate within different cellular compartments to prevent oxidative damage. Vitamin E is a lipid-soluble antioxidant located within cell membranes, where it scavenges free radicals directly. Selenium, as a cofactor for cytosolic glutathione peroxidase, destroys peroxides before they can attack membrane lipids, providing a complementary defense system.

61. (A) Massive eosinophilic infiltration
Skeletal muscle in White Muscle Disease is characterized by segmentary hyaline necrosis, myofiber mineralization, and infiltration of macrophages to clear necrotic debris, accompanied by satellite cell regeneration. However, massive eosinophilic infiltration is not a feature of this nutritional myopathy; instead, eosinophils are typical of parasitic or allergic reactions, which have a different etiology.

NODIA

62. (C) Microangiopathy and thrombosis
The pathogenesis of Mulberry Heart Disease involves severe oxidative damage to the microvasculature. A deficiency in Vitamin E or Selenium allows reactive oxygen species to damage capillary endothelial cells in the myocardium, leading to microangiopathy, fibrinoid degeneration, and microthrombosis. The resulting ischemia causes multifocal myocardial necrosis and extensive, confluent hemorrhages throughout the heart.

63. (D) I and II
White Muscle Disease and Exudative Diathesis are caused by deficiencies in Vitamin E and Selenium, which lead to free radical damage in muscle and capillaries, respectively. In contrast, Swayback is a neurological disorder characterized by demyelination and cavitation of the brain and spinal cord in lambs, which is caused by copper deficiency, not Vitamin E or Selenium.

64. (C) Thiamine
Polioencephalomalacia in ruminants is a neurological disease primarily associated with a deficiency of thiamine (Vitamin B1). Thiamine is a vital cofactor for key enzymes in the citric acid cycle and pentose phosphate pathway. Its deficiency disrupts cerebral energy metabolism, leading to cell swelling, ischemia, and subsequent necrosis of the gray matter of the cerebral cortex.

65. (A) Both A and R are true and R is the correct explanation of A.
Bracken fern contains active plant thiaminases, which are enzymes that chemically cleave and destroy dietary thiamine within the digestive tract. The loss of thiamine deprives neurons of a critical cofactor required for glucose metabolism and energy production. This metabolic arrest leads to neuronal energy failure, cellular swelling, and the development of cerebrocortical necrosis in affected animals.

calcium levels via calcium-sensing receptors, triggering continuous secretion of parathyroid hormone. Over time, this constant stimulation induces cellular proliferation, leading to diffuse bilateral hyperplasia of the parathyroid glands.

86. (D) Fibrous tissue replacement of bone
The hallmark microscopic lesion of osteodystrophia fibrosa is the progressive resorption of bone and its subsequent replacement by cellular fibrous connective tissue. Under the influence of elevated parathyroid hormone, osteoclasts actively degrade both cortical and cancellous bone. Osteoblasts and fibroblasts proliferate, filling the resulting marrow spaces and resorbed cavities with disorganized fibrous tissue.

87. (B) Osteodystrophia fibrosa
Osteodystrophia fibrosa is characterized by rapid resorption of bone, particularly the cancellous bone of the maxilla and mandible, and its replacement with fibrous tissue. This weak, proliferative tissue cannot withstand mechanical stress, leading to osteoid expansion and bilateral facial swelling. This presentation is classic in animals suffering from secondary hyperparathyroidism.

88. (C) Osteoclastic bone resorption
In the pathogenesis of osteodystrophia fibrosa, elevated levels of parathyroid hormone bind to receptors on osteoblasts, upregulating the expression of RANKL. RANKL binds to receptors on osteoclast precursors, stimulating their differentiation and activation. This induces intense osteoclastic bone resorption, rapidly degrading the mineralized bone matrix to release calcium into the circulation.

89. (D) Diffuse hyperplasia vs autonomous adenoma
Nutritional secondary hyperparathyroidism is distinguished by diffuse, bilateral hyperplasia of all parathyroid glands in response to chronic hypocalcemia. In contrast, primary hyperparathyroidism is caused by an autonomous, functional parathyroid adenoma or carcinoma that secretes parathyroid hormone independently of blood calcium. This primary neoplasm is typically unilateral, with the remaining glands appearing normal or atrophied.

90. (A) III only
The statement regarding renal calcium excretion is incorrect because parathyroid hormone actually promotes renal calcium reabsorption in the distal tubules, minimizing urinary loss of calcium. Simultaneously, it increases renal excretion of phosphorus. The other statements accurately describe how high phosphorus diets induce hypocalcemia, stimulating parathyroid hormone release and leading to bone resorption and fibrous osteodystrophy.

91. (B) Enzootic ataxia
Copper deficiency in pregnant ewes directly deprives the developing fetus of copper, leading to neonatal swayback or delayed-onset enzootic ataxia in lambs. Copper is a vital cofactor for enzymes like cytochrome c oxidase, which is required for myelin synthesis and maintenance. Its deficiency causes progressive demyelination and neuronal degeneration in the central nervous system.

92. (B) Cytochrome c oxidase
The demyelinating lesions of swayback are attributed to the functional failure of cytochrome c oxidase, a copper-dependent enzyme in the mitochondrial respiratory chain. In copper-deficient lambs, the lack of this enzyme impairs mitochondrial aerobic respiration, leading to energy failure in oligodendrocytes. This metabolic compromise disrupts the synthesis and maintenance of myelin, causing demyelination and cavitation.

93. (A) Tyrosinase
Achromotrichia, or loss of hair or wool pigmentation, in copper-deficient animals is caused by the impairment of tyrosinase. Tyrosinase is a copper-dependent enzyme essential for the biochemical pathway that converts tyrosine into melanin within melanocytes. Without adequate copper, tyrosinase activity is lost, halting melanin production and resulting in a pale or bleached coat.

94. (C) Lysyl oxidase
Lysyl oxidase is a copper-dependent extracellular enzyme that catalyzes the oxidative deamination of lysine residues in collagen and elastin. This enzymatic modification is essential for forming covalent cross-links that stabilize the extracellular matrix. In copper deficiency, lysyl oxidase activity is impaired, leading to weak connective tissue and spontaneous cardiovascular ruptures under arterial pressure.

Click Here to Buy book on Flipkart

depleted coagulation factors and culminating in fatal widespread internal hemorrhages.

76. (B) Inability to recycle oxidized vitamin K
The inhibition of vitamin K epoxide reductase prevents the conversion of oxidized vitamin K epoxide back into its active, reduced hydroquinone form. Without this recycling pathway, the cell rapidly exhausts its pool of active Vitamin K. This depletion prevents the gamma-carboxylation of crucial clotting proteins, disrupting the coagulation cascade and causing severe bleeding.

77. (A) Generalized hemorrhagic diathesis
The hallmark clinical pathological manifestation of severe Vitamin K deficiency or antagonism is a generalized hemorrhagic diathesis. Because multiple critical clotting factors are inactive, the primary and secondary coagulation pathways are severely impaired. This manifests as widespread, uncontrolled bleeding, including large subcutaneous hematomas, hemarthrosis, epistaxis, and internal hemorrhages into thoracic or abdominal cavities.

78. (C) Gamma-carboxylation of glutamic acid
Vitamin K functions as an essential cofactor for the enzyme gamma-glutamyl carboxylase, which catalyzes the post-translational gamma-carboxylation of glutamic acid residues on Factors II, VII, IX, and X. This chemical modification introduces negative charges, allowing these proteins to bind calcium and attach to phospholipid membranes on activated platelets, a crucial step in coagulation.

79. (D) Factor II (Prothrombin) has the shortest half-life and depletes first.
Factor VII, not Factor II, has the shortest biological half-life among the Vitamin K-dependent clotting factors and is depleted first during antagonism. This initial depletion rapidly prolongs the prothrombin time. In contrast, Factor II has the longest half-life and is the last to deplete, making the pairing of Factor II with the shortest half-life incorrect.

80. (C) Inhibition of vitamin K epoxide reductase
Second-generation anticoagulant rodenticides, such as brodifacoum, are highly potent and persistent toxins. They cause life-threatening hemorrhages by binding to and inhibiting vitamin K epoxide reductase in the liver. This block halts the recycling of oxidized Vitamin K, preventing the necessary activation of clotting factors and leading to severe, fatal coagulopathy in poisoned animals.

81. (B) Thrombocytosis
Sweet clover poisoning causes severe hemorrhage due to inactive Vitamin K-dependent clotting factors, manifesting as hematomas, hemarthrosis, and melena. However, thrombocytosis, an abnormally high platelet count, is not characteristic of this condition. While platelets are consumed during hemorrhages, the primary defect is a coagulopathy rather than a disorder of platelet production or hyperplasia.

NODIA

82. (D) High Phosphorus, Low Calcium
Nutritional secondary hyperparathyroidism is classically induced by diets with a low calcium content or an excessively high phosphorus-to-calcium ratio. High dietary phosphorus binds calcium in the intestinal lumen, preventing its absorption and causing transient hypocalcemia. This persistent drop in blood calcium stimulates the parathyroid glands to secrete excess parathyroid hormone, inducing bone resorption.

83. (A) Horses
Bran disease is a classic form of nutritional secondary hyperparathyroidism traditionally observed in horses fed diets high in phosphorus and low in calcium, such as wheat bran. The chronic mineral imbalance triggers continuous parathyroid hormone secretion, leading to osteodystrophia fibrosa. This bone resorption is most pronounced in the mandible and maxilla, causing swollen, distorted facial bones.

84. (C) P-2, Q-1, R-4, S-3
Bran disease describes facial osteodystrophia fibrosa in horses. Rubber jaw syndrome is seen in dogs with severe renal osteodystrophy. Big head disease refers to goats or sheep with facial bone swelling due to fibrous osteodystrophy. Rickets refers to defective mineralization in young, growing animals. Each condition arises from abnormal mineral homeostasis disrupting skeletal remodeling.

85. (A) Chronic hypocalcemia
The direct physiological stimulus for parathyroid gland hyperplasia in nutritional secondary hyperparathyroidism is chronic hypocalcemia. The parathyroid chief cells sense low extracellular

105. (D) Propionate
Ruminants ferment carbohydrates into volatile fatty acids, with propionate being the principal substrate for gluconeogenesis. The metabolism of propionate requires Vitamin B12 as a cofactor for the mitochondrial enzyme methylmalonyl-CoA mutase. A deficiency in Vitamin B12 halts this pathway, preventing the conversion of propionate to succinate and causing severe energy starvation.

106. (A) Hepcidin upregulates iron absorption during deficiency.
Hepcidin is a key hormone that downregulates iron absorption and release from macrophages. During iron deficiency, hepcidin expression is suppressed to facilitate increased dietary iron uptake. Conversely, high iron levels or systemic inflammation trigger increased hepcidin production to limit circulating iron levels, making its upregulation during deficiency physiological nonsense.

107. (B) Lack of dietary iron → Depletion of iron stores → Reduced hemoglobin synthesis → Microcytic hypochromic anemia
Rapidly growing piglets require substantial iron, which is deficient in sow milk and natural indoor environments. The progression to clinical disease initiates with this dietary lack, leading first to the exhaustion of limited neonatal hepatic stores. Consequently, hemoglobin synthesis declines due to insufficient iron availability, culminating in the classic morphological state of microcytic hypochromic anemia.

108. (B) Epidermal parakeratosis
Zinc is a vital cofactor for numerous metalloenzymes essential for cellular replication and protein synthesis within the integument. Insufficient zinc impairs normal epidermal maturation, leading to incomplete keratinization. This metabolic failure manifests grossly as thick, crusty skin scales and microscopically as epidermal parakeratosis, particularly affecting growing swine and certain canine breeds.

109. (A) Retention of nuclei in the stratum corneum
Epidermal parakeratosis is characterized histologically by the persistence of pyknotic nuclei within the cells of the stratum corneum. Under normal physiological conditions, keratinocytes lose their nuclei as they transition into this outermost layer. The retention of these nuclear remnants indicates a state of accelerated or abnormal cornification, commonly seen in nutritional zinc deficiency.

110. (C) II and IV
Iodine deficiency impairs the synthesis of thyroid hormones, leading to decreased circulating levels of thyroxine rather than an increase. This reduction removes the negative feedback on the pituitary gland, which causes an elevation in thyroid-stimulating hormone levels instead of suppression. Elevated thyroid-stimulating hormone subsequently drives follicular cell hyperplasia, resulting in goiter.

NODIA

111. (D) Thyroid-stimulating hormone
Inadequate iodine intake restricts the thyroid gland from synthesizing sufficient thyroid hormones. The resulting low concentrations of circulating thyroxine trigger the pituitary gland to release massive amounts of thyroid-stimulating hormone. This prolonged hormonal stimulation forces the thyroid follicular cells to undergo marked hypertrophy and hyperplasia, culminating in the macroscopically visible enlargement known as goiter.

112. (C) Follicles lined by tall columnar cells with minimal colloid
Chronic stimulation of the thyroid gland by thyroid-stimulating hormone causes follicular epithelial cells to become highly active, transforming from flat cuboidal cells to tall columnar cells. Because thyroid hormone synthesis is severely impaired, these hyperactive cells rapidly deplete the colloid stores, leaving the follicular lumens small, collapsed, and containing only minimal, pale-staining colloid.

113. (B) Inhibiting iodine uptake or organic binding by thyroid tissue
Goitrogenic substances present in plants like canola and kale disrupt the normal pathways of thyroid hormone synthesis. These active compounds interfere with the active transport of iodine into follicular cells or inhibit the essential iodination of tyrosine residues on thyroglobulin. This inhibition reduces thyroid hormone production, triggering compensatory pituitary release of thyroid-stimulating hormone.

114. (D) Calcium
Excess dietary calcium is a well-established antagonist of zinc absorption within the

which remains structurally intact and capable of secreting parathyroid hormone. Instead, the disorder stems from a temporary target-tissue insensitivity to parathyroid hormone, often induced by systemic metabolic alkalosis, combined with the sudden, overwhelming calcium demands of lactation.

134. (D) High dietary magnesium intake pre-partum
High pre-partum calcium and high dietary cation-anion difference diets reduce the tissue sensitivity to parathyroid hormone, predisposing cows to milk fever. Conversely, adequate or elevated dietary magnesium intake pre-partum actually protects against hypocalcemia. Magnesium is a vital cofactor for adenylate cyclase, which is necessary for parathyroid hormone synthesis and receptor signaling.

135. (B) Hypocalcemia, hypophosphatemia, and hypermagnesemia
Acute milk fever is biochemically characterized by a severe drop in blood calcium alongside secondary hypophosphatemia. The low phosphorus levels occur because elevated parathyroid hormone levels increase urinary and salivary phosphorus excretion. Additionally, cows commonly display hypermagnesemia because hypocalcemia reduces renal clearance of magnesium and impairs cellular uptake of this mineral.

136. (C) Sluggish and unresponsive
High dietary calcium intake during the dry period keeps blood calcium levels elevated, which continuously suppresses the parathyroid gland. Consequently, parathyroid hormone secretion and active vitamin D activation are minimal, rendering the homeostatic bone-mobilization systems inactive. At calving, the sluggish gland cannot adapt quickly enough to the sudden, massive calcium drain of lactation.

137. (D) Hindlimb ischemic crush myopathy
When a heavy dairy cow remains recumbent on hard surfaces, her immense body weight compresses the muscles and nerves of the down limbs. This compression compromises local microvasculature, leading to ischemia, venous congestion, and eventual tissue necrosis. This secondary damage, known as ischemic crush myopathy, prevents the cow from rising even after hypocalcemia is corrected.

138. (A) Loss of the swallowing reflex and silent regurgitation
Severe hypocalcemia induces flaccid paralysis of pharyngeal and esophageal smooth and skeletal muscle, which abolishes the protective swallowing reflex. Recumbent cows frequently experience silent regurgitation of acidic, microbially-rich ruminal contents. Without active pharyngeal control, this material is easily drawn down the trachea into the lungs, inducing highly destructive and often fatal aspiration pneumonia.

139. (B) Local compression of vasculature by high pressure
The heavy body mass of a recumbent cow exerts substantial physical pressure on the lowermost muscles, particularly the semitendinosus and semimembranosus. This pressure exceeds the normal capillary perfusion pressure, collapsing the local venous and arterial networks. The resulting lack of oxygen and nutrient delivery leads directly to localized ischemic muscle necrosis and swelling.

140. (B) Creatine kinase (CK)
Creatine kinase is an enzyme highly concentrated in skeletal and cardiac muscle tissues. When myocytes undergo necrosis or membrane damage during ischemic compression, creatine kinase leaks rapidly into the bloodstream. A dramatic rise in serum creatine kinase activity serves as a highly specific and sensitive indicator of active skeletal muscle damage and crush myopathy.

141. (A) Mastitis : Complete failure of milk synthesis
The increased incidence of mastitis in hypocalcemic cows is not due to a failure of milk synthesis, which actually continues. Instead, it is caused by flaccid paralysis of the teat sphincter muscle, which remains open and allows opportunistic pathogens to enter. Additionally, elevated cortisol levels and impaired immune responses further reduce the mammary defense systems.

142. (C) Coma or severe flaccid paresis
A blood calcium level of 4.5 mg/dL represents extremely severe hypocalcemia. At this critically low concentration, neuromuscular transmission is almost completely blocked because acetylcholine cannot be released at the motor endplate. The clinical presentation is dominated by complete flaccid paresis, lateral recumbency, loss of

Downloaded from www.pdf.tube

This massive redirection of vital substrates is the core trigger for metabolic transition failures.

124. (B) Insulin : Elevates during early lactation to conserve fat
Insulin concentrations actually drop dramatically during early lactation. This physiological decrease is essential to reduce glucose uptake by peripheral tissue, thereby sparing glucose for lactose synthesis in the mammary gland. Low insulin also facilitates the mobilization of adipose tissue reserves, allowing the cow to utilize non-esterified fatty acids as a primary systemic energy source.

125. (A) Bovine viral diarrhea
Production diseases are non-infectious metabolic imbalances directly linked to intensive high-yield animal farming. Conditions such as milk fever, ketosis, and post-parturient hemoglobinuria are classic examples because they stem from nutritional or metabolic shortfalls. Conversely, bovine viral diarrhea is an infectious, viral disease caused by a specific pestivirus, not a metabolic production breakdown.

126. (C) Flaccid paralysis
While hypocalcemia causes tetany in carnivores and horses, it produces flaccid paralysis in cows. This unique species difference exists because calcium is essential for the presynaptic release of acetylcholine at the neuromuscular junction. In cows, severe extracellular hypocalcemia blocks this neurotransmitter release, preventing muscle stimulation and resulting in generalized muscle weakness and recumbency.

127. (D) It triggers synaptic vesicle fusion and acetylcholine release.
Depolarization of the presynaptic nerve terminal opens voltage-gated calcium channels, prompting calcium influx. This influx is critical for triggering the fusion of synaptic vesicles with the presynaptic membrane, which releases acetylcholine into the synaptic cleft. In milk fever, depleted extracellular calcium prevents this entry, blocking neurotransmission and causing muscle paralysis.

128. (B) III, I, II
Parturient paresis progresses through three distinct, chronological clinical stages. Stage one is characterized by brief hyperexcitability, mild muscle tremors, and ataxia. As blood calcium drops further, stage two manifests as sternal recumbency, depression, and a characteristic S-shaped neck curve. Finally, stage three leads to lateral recumbency, coma, and death if untreated.

129. (B) Failure of acetylcholine release at the endplate
The primary neuromuscular defect in bovine hypocalcemia is the failure to release acetylcholine from the presynaptic nerve terminal. Because calcium ions are required to catalyze vesicle fusion and neurotransmitter exocytosis, depleted extracellular calcium levels interrupt the transmission of action potentials across the synaptic cleft, leaving skeletal muscles without the stimulation needed to contract.

130. (D) Sternal recumbency with the head tucked into the flank
During stage two of clinical parturient paresis, the affected cow is unable to stand and remains in sternal recumbency. Depressed consciousness and severe muscular weakness prevent her from holding her head upright. Consequently, she classically rests her muzzle against her flank, creating a highly characteristic tucked-head posture that is easily recognized during clinical examination.

NODIA

131. (A) Ionized calcium
Total blood calcium consists of protein-bound, complexed, and ionized fractions. The ionized calcium fraction represents the free, unbound form that is biologically active and directly responsible for neuromuscular transmission, membrane stability, and cardiac function. In milk fever, it is the acute depletion of this specific ionized pool that triggers clinical paresis.

132. (C) Gastrointestinal smooth muscle
Hypocalcemia impairs the contractility of smooth muscle as well as skeletal muscle. Paralysis of the gastrointestinal smooth muscle results in complete rumen stasis and abomasal atony. This cessation of motility stops the transit of digesta, halting the absorption of dietary calcium from the gut lumen and exacerbating the pre-existing systemic calcium deficit.

133. (A) It involves a primary defect in parathyroid gland structure.
Milk fever is not caused by a structural defect or primary disease of the parathyroid gland,

gastrointestinal tract of monogastric animals. High calcium levels promote the formation of insoluble complexes with zinc and phytic acid, making zinc unavailable for transport across the intestinal mucosa. This secondary mineral antagonism precipitates clinical parakeratosis despite apparently adequate dietary zinc concentrations.

115. (A) Severe exophthalmos
Iodine deficiency leads directly to hypothyroidism, which presents clinically as myxedema, alopecia, weak or stillborn neonates, and prolonged gestation in dams. Conversely, exophthalmos is a classic feature of hyperthyroidism or Graves-like disease in other species, where retro-orbital tissues swell due to immune activity or excess thyroid hormone, not thyroid hormone deficiency.

116. (C) Severe hypochromic macrocytic anemia
Zinc deficiency severely impairs cell division and immune functions, leading to clinical lesions such as thymic hypoplasia, delayed wound healing, and parakeratotic hyperkeratosis of the epidermis. However, severe hypochromic macrocytic anemia is not a pathological feature of zinc deficiency, as red blood cell production is primarily affected by iron, copper, or cobalt deficiencies.

117. (A) High production demands exceeding animal homeostatic capacity
Production diseases represent a modern veterinary challenge where high-yielding animals fail to maintain physiological equilibrium. Under intense production pressures, such as rapid onset of copious lactation, the high nutrient export exceeds the dam's homeostatic and adaptive regulatory mechanisms. This imbalance results in systemic metabolic collapses, which are exemplified by ketosis, hypocalcemia, and pregnancy toxemia.

118. (D) Homeorhesis
Homeorhesis is the coordinated regulation of bodily metabolism to support a dominant physiological state, such as lactation or rapid fetal growth. Unlike homeostasis, which acts to keep internal parameters constant, homeorhetic mechanisms orchestrate the systemic mobilization of lipid, protein, and mineral reserves from maternal tissues, prioritizing milk production even when it compromises maternal health.

119. (B) I, II, III, and V
Production diseases in dairy cows are highly concentrated in the early post-partum transition period rather than mid-lactation. Intense genetic selection and lagging dry matter intake during early lactation create a severe negative energy balance, driving fat mobilization and clinical metabolic disorders. Consequently, diseases like hypocalcemia and ketosis peak during these first few weeks of milk production.

120. (C) Non-esterified fatty acids mobilized from adipose tissues
Early lactation creates an extreme energetic deficit because the dietary intake of the cow is insufficient to cover mammary outputs. In response, homeorhetic signals stimulate intense lipolysis in adipose tissue, releasing vast amounts of non-esterified fatty acids into the circulation. These fatty acids travel to the liver to be oxidized, esterified, or converted into ketone bodies.

121. (D) Dynamic alterations in serum metabolite and mineral profiles
The transition from pregnancy to lactation forces rapid changes in mineral and energy demands. When a cow's adaptive systems fail to meet these demands, the breakdown is clearly reflected in abnormal blood parameters. Specifically, dramatic drops in serum calcium, increases in non-esterified fatty acids, and elevated ketone body concentrations serve as direct biochemical markers of homeostatic failure.

122. (A) Cortisol
The physiological stress of calving, paired with metabolic transition, triggers a marked activation of the hypothalamic-pituitary-adrenal axis. This activation results in significantly elevated levels of circulating cortisol around parturition. Cortisol exerts broad immunosuppressive effects on neutrophil function, lymphocyte blastogenesis, and cytokine production, predisposing periparturient dairy cows to severe infectious diseases like mastitis and metritis.

123. (B) Mammary gland
The initiation of lactation demands an unprecedented supply of glucose, amino acids, calcium, and lipids. At calving, the mammary gland becomes the dominant metabolic sink, pulling nutrients out of the bloodstream at a rate far exceeding the rate of digestive absorption.

Click Here to Buy book on Amazon

consciousness or deep coma, and impending death.

143. (D) Hepating lipidosis
While hypocalcemia directly impairs smooth muscle contractility—leading to uterine inertia, retained fetal membranes, dystocia, and reduced teat sphincter closure which causes mastitis—it does not directly cause hepatic lipidosis. Hepatic lipidosis is primarily a consequence of severe negative energy balance and rapid mobilization of adipose tissue, which is biochemically distinct.

144. (C) Severe negative energy balance
The fundamental driver of bovine ketosis is a severe negative energy balance occurring in early lactation. The cow's genetic drive for high milk production outstrips her physical capacity to ingest feed, resulting in an energy deficit. This deficiency triggers systemic lipolysis to meet energy demands, flooding the liver with non-esterified fatty acids that undergo incomplete oxidation.

145. (B) Oxaloacetate
Ruminants must synthesize almost all of their glucose via hepatic gluconeogenesis. When the mammary gland demands massive amounts of glucose for lactose synthesis, the liver prioritizes gluconeogenesis, heavily depleting the intracellular pool of oxaloacetate. Lacking sufficient oxaloacetate, acetyl-CoA cannot enter the tricarboxylic acid cycle, forcing it to be shunted into the pathway of ketogenesis.

146. (D) Propionate
Ruminants absorb very little glucose directly from their diet, as dietary carbohydrates are fermented in the rumen. Instead, they rely on propionate, the only major volatile fatty acid that is glucogenic. Absorbed propionate is transported to the liver, where it enters the gluconeogenic pathway to provide the vast majority of the animal's glucose.

147. (A) NEFA influx exceeds liver oxidation, causing ketogenesis and fat storage.
During negative energy balance, the rapid mobilization of adipose tissue floods the liver with non-esterified fatty acids. Because the capacity of the liver to oxidize these fatty acids or export them as very-low-density lipoproteins is limited, the excess fatty acids are re-esterified into triglycerides and stored within hepatocytes, while surplus acetyl-CoA is converted into ketones.

148. (C) Acetoacetate, beta-hydroxybutyrate, and acetone
When the liver's capacity to oxidize acetyl-CoA in the citric acid cycle is overwhelmed, the excess acetyl-CoA is directed toward ketogenesis. This pathway produces acetoacetate, which can be reversibly reduced to beta-hydroxybutyrate or spontaneously decarboxylated into acetone. These three chemical species constitute the major ketone bodies that accumulate in tissues during clinical acetonemia.

149. (A) Direct neurotoxic effects of high ketones paired with neuroglycopenia
Nervous ketosis is an acute neurological form of acetonemia characterized by delirium, head pressing, and compulsive licking. This syndrome is driven by a critical lack of glucose in the brain combined with the direct neurotoxic effects of extremely high concentrations of circulating acetoacetate and isopropyl alcohol, which is a breakdown product of accumulated acetoacetate and acetone.

150. (D) Excess Acetyl-CoA is converted entirely to glucose.
Acetyl-CoA cannot be converted into glucose in animal tissues because the pyruvate dehydrogenase reaction is irreversible and animals lack the glyoxylate cycle enzymes. Instead, during periods of low oxaloacetate, excess acetyl-CoA is shunted exclusively into the ketogenic pathway. Describing acetyl-CoA as a direct precursor for complete glucose synthesis is biochemically incorrect.

NODIA

151. (B) Acetate
Ruminant hepatic gluconeogenesis utilizes several substrates, including propionate, glucogenic amino acids, glycerol, and lactate. However, acetate is a two-carbon volatile fatty acid that is converted directly into acetyl-CoA. Because acetyl-CoA cannot be net-converted into glucose in animal cells, acetate serves only as an oxidative fuel or a precursor for fatty acid synthesis, never for gluconeogenesis.

152. (C) Acetone
Acetone is a volatile ketone body produced by the spontaneous, non-enzymatic decarboxylation of acetoacetate in the blood. Because of its

vital tissues of oxygen, causing severe anemic anoxia. Simultaneously, the large load of filtered hemoglobin causes tubular obstruction and nephrosis, leading to acute renal failure and cardiovascular collapse.

****

Downloaded from www.pdf.tube

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

162. (D) Competitively antagonize calcium to regulate acetylcholine release
Magnesium plays a crucial inhibitory role at the neuromuscular junction by acting as a natural, competitive antagonist to calcium. By competing for entry through presynaptic voltage-gated channels, magnesium limits the calcium influx required to trigger acetylcholine release. This physiological antagonism dampens motor nerve excitability, preventing unregulated, excessive skeletal muscle contraction and tetany.

163. (A) Cerebrospinal fluid
While blood serum magnesium levels can fluctuate rapidly, the onset of hypomagnesemic tetany correlates most closely with magnesium depletion in the cerebrospinal fluid. Because magnesium must cross the blood-brain barrier via active transport, cerebrospinal fluid concentrations represent the actual microenvironment of central neurons, where low magnesium directly causes hyperexcitability and tetanic spasms.

164. (C) Potassium and Nitrogen
Lush, rapidly growing spring pastures are frequently high in potassium and nitrogen due to heavy fertilization. High potassium levels in the rumen fluid directly depolarize the apical membrane of ruminal epithelial cells, blocking the active, electrogenic transport of magnesium. High nitrogen levels further reduce magnesium absorption by increasing ruminal ammonia concentrations, which precipitates magnesium.

165. (A) Excessive release of acetylcholine at the motor endplate
In hypomagnesemic states, the loss of competitive inhibition by magnesium allows calcium ions to enter the presynaptic nerve terminal in an unregulated manner. This excessive calcium entry triggers continuous and massive exocytosis of acetylcholine into the synaptic cleft. The resulting persistent stimulation of postsynaptic receptors leads directly to clinical muscle twitching, hyperexcitability, and tetanic spasms.

166. (D) Vitreous humor or CSF
Following death, autolysis rapidly alters blood serum mineral concentrations, making post-mortem blood samples highly unreliable for diagnosing hypomagnesemia. In contrast, the vitreous humor of the eye and the cerebrospinal fluid are anatomically isolated and remain biochemically stable for up to twenty-four to forty-eight hours post-mortem, making them the most reliable diagnostic samples.

167. (B) They lack a mobilizable hormonal reserve of magnesium in bone.
Ruminants are highly sensitive to a sudden drop in dietary magnesium because they lack a rapidly mobilizable body pool of this mineral. While skeletal tissues contain large amounts of magnesium, it is tightly bound within the crystalline bone matrix and cannot be rapidly resorbed. Consequently, adult ruminants depend on constant, daily dietary absorption to maintain physiological blood concentrations.

168. (C) Parathyroid hormone rapidly mobilizes magnesium from adult bones.
While parathyroid hormone regulates calcium homeostasis by rapidly mobilizing calcium from skeletal tissues, it has no direct capacity to stimulate the rapid resorption of magnesium from adult bones. Bone magnesium in adults is structurally locked and highly inert. Because there is no major hormonal homeostatic system for mobilizing magnesium, blood levels depend entirely on daily dietary intake.

NODIA

169. (D) High intake of mature, dry hay
Hypomagnesemic tetany is precipitated by factors that limit magnesium intake or impair its ruminal absorption. Examples include grazing lush spring pastures high in potassium, transport stress, and cold, wet weather which reduces grazing activity. Conversely, feeding muscle, dry hay provides structural fiber and has lower potassium concentrations, which actually promotes stable and efficient magnesium absorption.

170. (A) Streptococcus bovis
Ingestion of excessive non-structural carbohydrates provides an abundant, easily fermentable substrate in the rumen. This nutritional surge triggers the rapid multiplication of Streptococcus bovis, a highly active, starch-utilizing bacterium. Streptococcus bovis metabolizes carbohydrates into lactic acid, which rapidly lowers the ruminal pH and initiates the progressive destruction of the normal, cellulolytic microflora population.

2015

9. In toxicokinetics, Phase I biotransformation reactions primarily involve which of the following processes?
(A) Conjugation with glucuronic or sulfuric acid
(B) Active transport of metabolites into the bile
(C) Glomerular filtration through the kidney nephrons
(D) Oxidation, reduction, and hydrolysis reactions

2017

10. Which process best defines toxicokinetic distribution?
(A) The movement of an absorbed toxicant via blood to tissues
(B) The irreversible binding of a toxicant to hepatic enzymes
(C) The chemical alteration of a compound to a polar metabolite
(D) The elimination of a volatile toxicant through exhalation

2017

11. If a xenobiotic is highly lipophilic, what inference can be made about its toxicokinetics?
(A) It will undergo rapid filtration and be excreted unchanged in the urine.
(B) It will require Phase II conjugation prior to renal excretion.
(C) It will fail to penetrate or cross the blood-brain barrier.
(D) It will undergo active tubule secretion without lipid absorption.

2008

12. All of the following are primary routes of xenobiotic excretion in animals EXCEPT:
(A) Biliary excretion
(B) Splenic sequestration
(C) Renal excretion
(D) Pulmonary exhalation

2007

13. A dog ingests a toxin that acts as an enzyme inducer for cytochrome P450. How will this affect the application of a concurrently administered therapeutic drug metabolized by the same pathway?
(A) The therapeutic drug will be metabolized and excreted more rapidly.
(B) The therapeutic drug will accumulate in tissue to toxic levels.
(C) The therapeutic drug will bypass hepatic portal circulation entirely.
(D) The therapeutic drug's absorption from the small intestine will halt.

2014

14. Which statement correctly characterizes Phase II biotransformation?
(A) It is restricted to reactions that make the toxicant highly lipophilic.
(B) It is catalyzed and mediated exclusively by cytochrome P450 enzymes.
(C) It involves conjugation with polar molecules to increase solubility.
(D) It invariably increases the pathological toxicity of the xenobiotic.

2012

15. Consider the following statements regarding toxicokinetics:
I. Absorption in the stomach favors weak acids.
II. Enterohepatic recirculation accelerates the elimination of a toxicant.
III. Glucuronidation is a common Phase I reaction.
IV. The blood-brain barrier excludes highly lipid-soluble toxins.
Which of the above statements are INCORRECT?
(A) I and II
(B) II, III and IV
(C) I and III
(D) I, II and IV

2009

16. Compared to dermal absorption, inhalation of a toxic gas typically results in:
(A) A delayed onset and slower distribution of systemic clinical toxicity
(B) An increased deposition and accumulation within adipose tissue deposits
(C) Direct systemic circulation entry bypassing first-pass hepatic clearance
(D) An enhanced rate of compound elimination via biliary fecal excretion

2008

17. The primary site for xenobiotic biotransformation in animals is the:
(A) Kidney
(B) Liver
(C) Lung
(D) Spleen

2022

18. Oxidative stress in cells is primarily driven by an imbalance between the production of reactive oxygen species (ROS) and the presence of:
(A) Endonucleases
(B) Excitatory neurotransmitters

Click Here to Buy book on Flipkart

epithelial necrosis, inflammation, and chemical rumenitis.

181. (A) Hemorrhagic infarction and submucosal thrombosis
Fungi are highly angioinvasive organisms. When they colonize areas of chemical rumenitis, they penetrate the damaged mucosal layers and invade local blood vessels. This vascular invasion triggers extensive submucosal thrombosis, which cuts off blood flow and results in well-circumscribed, dark red, circular areas of hemorrhagic infarction within the ruminal wall.

182. (D) Mucor, Rhizopus, and Aspergillus
Mycotic rumenitis is primarily caused by opportunistic fungi that thrive on necrotic tissues and in acidic environments. The most common etiologies belong to the zygomycetes, specifically Mucor and Rhizopus, alongside Aspergillus species. These opportunistic molds readily invade the compromised ruminal wall, causing the classic angioinvasive lesions and thrombosis associated with this disease.

183. (B) Pulmonary artery aneurysm rupture secondary to caval thrombosis
Septic emboli from a caudal vena caval thrombus travel directly to the lungs, where they lodge in the pulmonary arteries and cause focal arteritis. This vascular inflammation weakens the arterial walls, leading to the formation of pulmonary aneurysms. The sudden rupture of these aneurysms into the bronchial tree causes massive, fatal airway hemorrhage and epistaxis.

184. (C) Bacteria travel via the hepatic vein to colonize the liver.
In the pathogenesis of the rumenitis-liver abscess complex, bacteria that breach the damaged ruminal mucosa enter the portal vein, which carries venous blood from the gastrointestinal tract directly into the liver. They do not travel via the hepatic vein, which actually drains blood away from the liver toward the caudal vena cava.

185. (D) Mycobacterium bovis
The rumenitis-liver abscess complex is caused by opportunistic rumen bacteria that translocate via the portal circulation, including Fusobacterium necrophorum, Trueperella pyogenes, and Bacteroides species. Conversely, Mycobacterium bovis is the causative agent of bovine tuberculosis, which is a chronic granulomatous disease transmitted primarily via respiratory aerosol, not an opportunistic post-acidosis pathogen.

186. (A) Lush legumes high in soluble leaf proteins
Primary ruminal tympany is triggered by the rapid digestion of young, succulent legumes such as alfalfa or clover. These plants are exceptionally rich in soluble leaf proteins. When fermented in the rumen, these proteins act as foaming agents, creating a stable, viscous foam that traps the gases of fermentation and prevents normal eructation.

187. (B) An esophageal "bloat line" with cranial congestion and caudal pallor
Severe ruminal distension exerts massive pressure on the thoracic cavity and diaphragm. This pressure blocks venous return from the head and neck, causing intense congestion and hemorrhage in the cranial esophagus. Conversely, the intrathoracic esophagus is compressed and appears pale. The sharp demarcation between these two zones is the pathognomonic bloat line.

188. (B) Asphyxiation and collapse from diaphragmatic compression
The massive, rapid accumulation of gas or foam causes extreme distension of the rumen, which pushes forward against the diaphragm. This compression restricts the movement of the lungs, severely reducing thoracic volume and preventing normal ventilation. Consequently, the animal dies of progressive asphyxiation, tissue hypoxia, and acute cardiovascular collapse.

189. (A) Failure of the eructation mechanism
Secondary ruminal tympany occurs when fermentation gases are produced normally but cannot be released. This free-gas accumulation is driven by a failure of the eructation mechanism, which can be caused by physical blockages in the esophagus, damage to the vagus nerve, or systemic conditions that inhibit normal reticulorumen motility.

190. (C) Vagal nerve injury or mediastinal tissue compression
Chronic secondary free-gas bloat is frequently caused by damage to the vagus nerve, which coordinates the complex muscular contractions required for eructation. Similarly, physical compression of the esophagus or vagal branches by enlarged mediastinal lymph nodes—often

Downloaded from www.pdf.tube

resulting from pneumonia or lymphoma—interferes with normal motility, leading to persistent gas accumulation.

191. (D) Plant proteins, saponins, and pectins
The formation of stable foam in primary bloat depends on specific chemical agents derived from ingested forage. Soluble plant proteins, particularly fraction one leaf proteins, act as powerful surfactants. When combined with other plant constituents like saponins and pectins, they create a highly cohesive, viscous film that traps gas bubbles within the ruminal fluid.

192. (C) The hindquarters are congested while the forequarters are pale.
In an animal that died of bloat, the massive intra-abdominal pressure compresses the caudal vena cava, restricting venous blood return from the hindquarters. This causes the hindlimbs to appear pale and ischemic. Conversely, the increased thoracic pressure forces venous blood to pool in the neck, head, and forequarters, which appear heavily congested.

193. (B) Ingestion of high-legume pasture
Secondary ruminal tympany is caused by physical or functional blockages that prevent free gas from being eructated, such as choke, tetanus, or reticular adhesions. In contrast, the ingestion of high-legume pastures triggers primary ruminal tympany, where gas is trapped in a stable foam rather than accumulating as free gas.

194. (D) Phosphorus
Post-parturient hemoglobinuria is a metabolic disease of high-producing dairy cows characterized by intravascular hemolysis. The underlying cause is a severe, acute deficiency of phosphorus. The onset of heavy lactation creates an enormous phosphorus drain into milk, depleting the extracellular phosphorus pool and impairing the metabolic stability of circulating erythrocytes.

195. (A) Massive acute intravascular hemolysis
The defining pathological event in post-parturient hemoglobinuria is massive, acute intravascular hemolysis. Because red blood cells are depleted of key energy molecules, they lose their structural integrity and rupture directly within the bloodstream. This systemic breakdown floods the plasma with free hemoglobin, leading to clinical hemoglobinemia, hemoglobinuria, and severe, life-threatening anemic hypoxia.

196. (C) Intracellular ATP and Na^+/K^+ pump function
Erythrocytes depend on glycolysis to generate adenosine triphosphate, which is required to power the sodium-potassium ATPase pump. Severe hypophosphatemia deprives these cells of inorganic phosphorus, halting glycolysis and depleting intracellular adenosine triphosphate. Consequently, the active pumps fail, leading to uncontrolled sodium accumulation, cellular swelling, and membrane lysis.

197. (A) Fragile spherocyte
Without sufficient adenosine triphosphate, erythrocytes cannot maintain the cytoskeletal integrity needed for their normal, flexible biconcave shape. The cells swell, lose portions of their membrane, and transform into fragile, rigid spherocytes. These abnormal cells are highly susceptible to mechanical damage in the microvasculature, leading to rapid intravascular hemolysis.

NODIA

198. (D) Two to four weeks following calving
Unlike milk fever, which occurs within hours of calving, post-parturient hemoglobinuria typically manifests two to four weeks post-partum. This period coincides with the peak of milk production, when the mammary excretion of phosphorus reaches its maximum. This sustained, high-volume loss eventually exhausts maternal body phosphorus reserves, precipitating the acute hemolytic crisis.

199. (B) Formation of Heinz bodies on erythrocytes
Post-parturient hemoglobinuria is characterized by a low packed cell volume, hemoglobinemia, and hemoglobinuria, which are caused by ATP depletion. In contrast, the formation of Heinz bodies is a hallmark of oxidative damage to hemoglobin, which is typically seen in onion poisoning, brassica toxicosis, or red maple leaf toxicity.

200. (C) The primary defect is a lack of renal erythropoietin production.
The pathogenesis of post-parturient hemoglobinuria is centered on metabolic phosphorus depletion, which impairs glycolysis and causes erythrocyte lysis. It does not involve a primary defect in renal erythropoietin

production, as the kidneys are capable of secreting erythropoietin. The anemia is hemolytic and regenerative, rather than a failure of bone marrow production.

201. (D) Immune-complex deposition on the membrane
Erythrocyte ATP depletion leads to sodium and water accumulation, loss of membrane deformability, and physical membrane rupture. However, immune-complex deposition does not occur, as this process is characteristic of immune-mediated hemolytic anemia. The destruction of red cells in post-parturient hemoglobinuria is purely metabolic and mechanical, rather than immune-driven.

202. (A) "Gunmetal kidneys"
During massive intravascular hemolysis, the blood plasma is flooded with free hemoglobin, which is filtered by the glomeruli. The renal tubules absorb excess hemoglobin, and the accumulation of this pigment within the tubular epithelium imparts a highly characteristic dark, metallic, red-to-black discoloration to the kidneys, a lesion commonly referred to as gunmetal kidneys.

203. (B) Severe anemic hypoxia
The acute, massive destruction of erythrocytes reduces the oxygen-carrying capacity of the blood, leading to systemic hypoxia. Because hepatocytes surrounding the central veins receive blood that is already depleted of oxygen, they are highly sensitive to oxygen deprivation. This hypoxemia results in severe, diffuse centrilobular hepatic necrosis in fatal cases.

204. (B) Pre-hepatic (hemolytic)
The rapid, widespread lysis of red blood cells in the vasculature releases enormous amounts of hemoglobin, which is metabolized into unconjugated bilirubin. This influx of bilirubin exceeds the conjugation and excretory capacity of the liver, leading to the accumulation of yellow pigment in tissues. This is classified as pre-hepatic or hemolytic icterus.

205. (A) Brightly eosinophilic hemoglobin casts in the tubules
Histological evaluation of kidneys from cows with severe hemoglobinuric nephrosis reveals extensive tubular damage. The lumens of the proximal and distal convoluted tubules are filled with abundant, brightly eosinophilic to orange-brown proteinaceous casts composed of precipitated free hemoglobin. This intratubular accumulation is accompanied by degeneration and necrosis of the lining epithelial cells.

206. (C) Hemosiderin
Following an episode of intravascular hemolysis, macrophages in the spleen, liver, and lymph nodes phagocytose the debris of damaged erythrocytes and free hemoglobin. The iron recovered from this process is stored intracellularly as hemosiderin. Consequently, tissues display a dense, golden-brown accumulation of hemosiderin pigment, which stains positive with Prussian blue.

207. (D) Hemoglobinuric urine remains uniformly red/brown after centrifugation.
Centrifugation of urine is the most reliable rapid method to differentiate hematuria from hemoglobinuria. In hematuria, intact red blood cells settle to the bottom, leaving a clear supernatant. In hemoglobinuria, the red-to-brown color is caused by free, dissolved hemoglobin, which does not settle, meaning the urine remains uniformly colored after centrifugation.

208. (C) Splenomegaly : Primary lymphoid neoplasia
The splenomegaly observed in cases of post-parturient hemoglobinuria is not caused by lymphoid neoplasia. Instead, it is a physiological hyperplastic response driven by the sudden, massive demand for phagocytosis of damaged erythrocytes and cellular debris by splenic macrophages. This is an active, reactive congestive and hemolytic process, not a neoplastic disease.

209. (B) Isosthenuria and positive occult blood tests without intact erythrocytes
Hemoglobinuric nephrosis causes severe acute kidney injury, leading to a loss of tubular concentrating ability, which manifests as isosthenuria. Because the urine is filled with dissolved free hemoglobin rather than intact cells, chemical test strips will indicate a strong positive result for blood, while microscopic evaluation of the sediment will reveal no intact erythrocytes.

210. (D) Severe anemic anoxia and acute renal failure
The terminal phase of post-parturient hemoglobinuria is characterized by a fatal combination of metabolic and organ failures. The massive loss of erythrocytes deprives

Click Here to Buy book on Amazon

volatility, accumulated acetone readily diffuses across the alveolar-capillary membrane into the airways. The pulmonary excretion of this chemical is directly responsible for the sweet, fruity, or solvent-like odor of the breath in ketotic animals.

153. (D) Rapid late-gestation fetal growth, especially with multiple fetuses
Ovine pregnancy toxemia is a metabolic disease of pregnant ewes that occurs during late gestation. Approximately eighty percent of fetal skeletal and tissue growth happens during the final six weeks of pregnancy, requiring massive amounts of maternal glucose. When carrying multiple fetuses, this glucose demand escalates, overwhelming the ewe's capacity and precipitating severe hypoglycemic crisis and ketosis.

154. (A) Neuroglycopenia
The central nervous system of sheep has an absolute requirement for glucose as its primary energy substrate. The extreme fetal demand in late gestation severely depletes maternal blood glucose. The resulting neuroglycopenia starves cerebral neurons of energy, impairing basic cellular functions and manifesting clinically as cortical blindness, depression, tremors, star-gazing, and eventual recumbency.

155. (B) Combined fetal glucose demands exceed the ewe's maximal gluconeogenic capacity.
Ewes carrying multiple fetuses face a critical physiological challenge in late pregnancy. The combined glucose demand of twin fetuses is extremely high, requiring more glucose than the maternal liver can produce through gluconeogenesis. When feed intake is physically restricted by the expanding gravid uterus, the ewe cannot meet these immense metabolic demands, initiating rapid clinical disease.

156. (B) A pale yellow, enlarged, greasy, and friable liver
In response to the extreme glucose deficit, the ewe mobilizes extensive lipid reserves from adipose depots. This massive influx of non-esterified fatty acids overwhelms the liver's processing capacity. On necropsy, this severe hepatic lipidosis is clearly visible, presenting as an enlarged, pale yellow, greasy, and easily torn liver that can even float in water.

157. (A) Astrocyte swelling and neuronal necrosis
Severe, prolonged hypoglycemia and ketonemia in late-term ewes disrupt the metabolic integrity of cerebral tissues. Histological examination of the brain reveals swollen astrocytes, which reflects a failure of cellular volume regulation and ion homeostasis. Additionally, ischemic neuronal necrosis or "red dead" neurons are observed within the cerebral cortex, explaining the irreversible neurological deficits.

158. (C) Cattle ketosis is driven by lactation, whereas sheep ketosis is driven by fetal growth.
Although both diseases involve severe negative energy balance, their physiological timing is different. Bovine ketosis is predominantly a post-partum production disease driven by the immense glucose demand of early lactation. In contrast, ovine pregnancy toxemia is a pre-partum metabolic disease driven by the exponential growth and energy requirements of multiple fetuses in late gestation.

NODIA

159. (D) Massive increases in circulating insulin
The metabolic crisis of pregnancy toxemia is characterized by hypoglycemia, which naturally triggers a physiological suppression of insulin secretion. Low insulin levels are necessary to permit maximum mobilization of energy reserves. Therefore, massive increases in circulating insulin do not occur; instead, insulin levels are severely decreased, while glucagon and cortisol concentrations are markedly elevated.

160. (C) BUN and creatinine
As pregnancy toxemia progresses, affected ewes cease eating and drinking, leading to severe dehydration, metabolic acidosis, and hypovolemic shock. This state of low systemic perfusion compromises renal blood flow, causing a dramatic drop in glomerular filtration. Consequently, nitrogenous waste products accumulate in the bloodstream, manifesting as markedly elevated serum urea nitrogen and creatinine.

161. (B) Massive fibrinopurulent peritonitis
Necropsy of an ewe that succumbed to pregnancy toxemia typically reveals an empty rumen, an enlarged, pale yellow, greasy fatty liver, and multiple late-term fetuses within the uterus. However, massive fibrinopurulent peritonitis is not a feature of this metabolic disease, as there is no primary bacterial infection or rupture of abdominal viscera.

CHAPTER 13
Pathology of Toxicosis

2008

1. Which of the following best defines a xenobiotic in veterinary pathology?
(A) A naturally occurring biological toxin synthesized by plants
(B) Any chemical substance foreign to the biological system of the animal
(C) An endogenous compound responsible for metabolic regulation
(D) A synthetic mineral supplement essential for bone development

2022

2. The term 'toxicosis' refers specifically to which of the following?
(A) The lethal dose of a specific poison
(B) The metabolic breakdown of a poison in the liver
(C) The accumulation of toxins within adipose tissue over time
(D) The pathological condition or disease state resulting from exposure to a toxicant

2018

3. Which of the following is an example of an endogenous toxicant?
(A) Ammonia generated from ruminal microbial degradation of urea
(B) Organophosphate insecticide
(C) Mycotoxin from contaminated feed
(D) Lead from industrial waste

2019

4. Which statement correctly identifies the distinction between a toxin and a toxicant?
(A) Toxins are synthetic, whereas toxicants are naturally produced.
(B) Toxins only affect mammals, whereas toxicants affect all species.
(C) Toxins are of biological origin, whereas toxicants include all poisonous substances.
(D) Toxins are organic poisons, whereas toxicants are heavy metals exclusively.

2004

5. Consider the following statements regarding toxicant classification:
I. Xenobiotics are exclusively man-made chemicals.
II. Venoms are specific types of toxins delivered via animal bite or sting.
III. Phytotoxins are toxic substances produced by plants.
Which of the above statements is/are correct?
(A) I and II
(B) II and III
(C) I only
(D) III only

2013

6. Which of the following pairs INCORRECTLY matches a term with its fundamental characteristic?
(A) Lethal Dose 50 (LD50) - Dose killing half the exposed population
(B) Teratogen - Chemical causing structural fetal abnormalities
(C) Carcinogen - Agent promoting malignant tumor formation
(D) Mutagen - Substance preventing cellular DNA mutations

2011

7. What is the primary cause of acute poisoning in veterinary cases compared to chronic toxicity?
(A) Prolonged, continuous exposure to low-level toxic substances
(B) A single exposure to a massive dose of a toxic agent
(C) Gradual accumulation of heavy metals in the bone tissue
(D) Genetic predisposition that alters metabolic enzyme pathways

2013

8. Based on their origin, how are mycotoxins appropriately classified?
(A) Synthetic agricultural toxicants
(B) Endogenous host metabolic poisons
(C) Fungal biological toxins
(D) Inorganic environmental xenobiotics

Downloaded from www.pdf.tube

171. (B) A shift from Gram-negative flora to lactic-acid-producing Gram-positive bacteria
The normal ruminal environment is dominated by Gram-negative cellulolytic bacteria and protozoa. As easily fermentable starch is introduced, the pH drops, killing these sensitive Gram-negative organisms. This acidic shift allows acid-tolerant, Gram-positive, lactic-acid-producing bacteria—specifically Streptococcus bovis and eventually Lactobacillus species—to dominate, accelerating the production of lactic acid in a self-reinforcing cycle.

172. (B) D-lactate
Ruminal fermentation of starch produces both L-lactate and D-lactate isomers. While animals possess abundant lactate dehydrogenase to rapidly metabolize the L-isomer, they have very limited pathways to process D-lactate. Consequently, absorbed D-lactate accumulates in the systemic circulation, causing profound, long-lasting metabolic acidosis, systemic dehydration, and progressive central nervous system depression.

173. (A) Elevated ruminal fluid osmolarity, drawing water from circulation into the rumen
The massive accumulation of lactic acid and glucose breakdown products inside the rumen increases the osmolarity of the ruminal fluid. This hyperosmotic state creates a strong osmotic gradient that draws large volumes of water from the vascular system and extracellular spaces across the ruminal wall, resulting in severe systemic dehydration and hemoconcentration.

174. (C) Death and lysis of ruminal protozoa
As the rumen pH falls below 5.5 due to initial lactic acid accumulation, the highly sensitive ciliated protozoa are rapidly destroyed. The death and lysis of these protozoa is one of the earliest biological events in grain overload. Their disappearance is easily detected during microscopic evaluation of ruminal fluid, serving as a reliable diagnostic indicator of acute acidosis.

175. (D) High anion gap metabolic acidosis
The extensive absorption of D-lactate and L-lactate from the rumen floods the bloodstream with unmeasured organic anions. These circulating lactate anions consume bicarbonate buffers, leading to a severe metabolic acidosis. Biochemically, this is characterized by a high anion gap, which reflects the accumulation of these unmeasured acid anions in the plasma.

176. (C) Increased ruminal pH
Grain overload is defined by a dramatic and dangerous drop in ruminal pH, often falling from a normal neutral range down to 4.5 or lower. This acidic environment is caused by the rapid bacterial fermentation of carbohydrates into lactic acid. Stating that grain overload results in an increased ruminal pH is a physiological contradiction.

177. (B) Intravascular hemolysis
Acute ruminal acidosis results in severe dehydration, oliguria, scleral congestion, and laminitis, which are driven by systemic lactic acidosis and vasoactive mediator release. However, intravascular hemolysis is not a direct consequence of this syndrome. Hemolysis is instead associated with conditions such as copper toxicity, water deprivation-sodium toxicosis, or post-parturient hemoglobinuria.

178. (D) Fusobacterium necrophorum
Chemical rumenitis breaches the protective mucosal barrier, allowing bacteria to enter the portal venous system. These bacteria travel directly to the liver parenchyma. Fusobacterium necrophorum, a Gram-negative obligate anaerobe that is part of the normal ruminal flora, is the primary pathogen that colonizes these sites, producing a highly destructive leukotoxin that results in necrobacillary hepatic abscesses.

179. (A) Caudal vena cava, causing thrombosis
Hepatic abscesses can expand and rupture through the walls of adjacent blood vessels. The most dangerous sequela occurs when an abscess erodes into the caudal vena cava, initiating septic thrombosis. This thrombosis can lead to embolization of the lungs, pulmonary arterial hypertension, aneurysm formation, and eventual fatal hemorrhage into the airways.

180. (C) Direct caustic action of un-dissociated lactic acid
When the ruminal pH drops significantly, lactic acid exists largely in its un-dissociated lipid-soluble form. This un-dissociated acid easily penetrates the cell membranes of the stratified squamous epithelium lining the rumen. Inside the cells, it dissociates and releases hydrogen ions, exerting a direct caustic effect that leads to

NODIA

(C) Pro-apoptotic proteins
(D) Antioxidant defenses

2016
19. Which definition best describes apoptosis as a mechanism of toxicant-induced cell death?
(A) Caspase-mediated programmed cell death with cellular fragmentation
(B) Unregulated swelling of cells leading to plasma membrane rupture
(C) Inflammatory dissolution of damaged cells mediated by neutrophils
(D) Progressive tissue calcification occurring under normal calcium levels

2018
20. Toxicant-induced depletion of cellular ATP directly leads to which immediate cellular effect?
(A) Upregulation and enhancement of ribosomal protein translation
(B) Prompt and immediate initiation of cellular division pathways
(C) Dysfunction of Na^+/K^+ ATPase pumps resulting in cellular swelling
(D) Accelerated transcription and replication of mitochondrial DNA

2014
21. All of the following are characteristic features of necrotic cell death in toxicosis EXCEPT:
(A) Intact cell membranes
(B) Eosinophilic cytoplasm
(C) Nuclear karyolysis
(D) Inflammatory response

2006
22. If a toxicant specifically targets and disrupts the mitochondrial inner membrane, it can be inferred that the cell will suffer from:
(A) Accelerated accumulation of undigested lysosomal storage
(B) Upregulated secretory transport of the Golgi apparatus
(C) Unregulated synthetic activity of hepatic glycogen stores
(D) Rapid failure of oxidative phosphorylation and ATP synthesis

2021
23. Which statement INCORRECTLY describes membrane damage during cell injury?
(A) Lipid peroxidation is triggered by free radicals attacking membrane lipids.
(B) Membrane damage exclusively affects the plasma membrane, sparing organelles.
(C) Phospholipase activation contributes to membrane breakdown.
(D) Cytoskeletal abnormalities can exacerbate membrane structural failure.

2006
24. What is a key disadvantage of utilizing purely morphological criteria to distinguish between apoptosis and necrosis in toxicological pathology?
(A) Apoptosis is only identifiable utilizing high-resolution electron microscopy systems.
(B) Necrosis fails to induce any detectable macroscopic or microscopic tissue alterations.
(C) Severe acute toxic insult often triggers overlapping features of both death pathways.
(D) Morphological differentiation is completely unreliable when applied to glandular organs.

2013
25. Consider the following statements concerning cell injury pathogenesis:
I. Calcium influx into the cytosol activates destructive enzymes.
II. Cytochrome c release from mitochondria triggers apoptosis.
III. Superoxide dismutase generates free radicals.
IV. Lipid peroxidation produces reactive aldehydes.
V. Glutathione acts as a major cellular antioxidant.
Which of the above statements are correct?
(A) I, II, III and V
(B) I, II, IV and V
(C) II, III and IV
(D) I, III, IV and V

2012
26. In an applied scenario where an animal presents with severe lipid peroxidation of hepatocyte membranes, administration of which agent would theoretically mitigate further injury?
(A) An iron chelator or antioxidant
(B) A highly lipophilic toxicant
(C) An inducer of cytochrome P450
(D) A competitive antagonist of acetylcholine

2019
27. Which host factor critically limits the ability of cats to metabolize certain phenolic toxicants like acetaminophen?
(A) Complete absence of functional ruminal microflora
(B) Insufficient level of glucuronyl transferase activity
(C) Excessive overexpression of hepatic Phase I enzymes

Click Here to Buy book on Flipkart

(C) Severe mucosal edema with intense "rose-red" hyperemic inflammation
(D) Complete atrophy and total absence of normal gastric mucosal folds

2016
47. Which statement INCORRECTLY characterizes arsenic toxicity?
(A) It is commonly found in herbicides and various wood preservatives.
(B) Chronic exposure leads to cutaneous lesions and hyperkeratosis.
(C) It induces profound systemic vasodilation and capillary leakage.
(D) It acts as a pure neurotoxin sparing the gastrointestinal tract.

2003
48. In a flock of sheep, several die rapidly with profuse watery diarrhea containing blood. Post-mortem shows intense abomasal hyperemia. Based on these applied signs, the pathologist must prioritize testing for:
(A) Organochlorine toxicity
(B) Fumonisin toxicity
(C) Arsenic toxicity
(D) Chronic copper toxicity

2006
49. If a tissue sample shows severe paralysis of capillary beds leading to exudation of plasma, one can infer the likely involvement of:
(A) Lead
(B) Arsenic
(C) Selenium
(D) Mercury

2003
50. Historically, arsenic was primarily used in veterinary medicine as a/an:
(A) Broad-spectrum antibiotic agent
(B) Highly specific antiviral agent
(C) Systemic anti-inflammatory drug
(D) Anthelmintic and growth promoter

2002
51. Which form of mercury is most highly absorbed across the blood-brain barrier and associated with severe neurotoxicity?
(A) Organic methylmercury
(B) Elemental liquid mercury
(C) Inorganic mercury salts
(D) Mercury sulfide

2018
52. The profound acute nephrotoxicity caused by inorganic mercury is a direct effect of its concentration in the:
(A) Renal glomerular capillary beds
(B) Renal pelvis transitional mucosa
(C) Proximal convoluted tubules
(D) Renal medullary collecting ducts

2017
53. Which statement correctly links the type of mercury with its primary target organ?
(A) Methylmercury primarily targets the central nervous system.
(B) Inorganic mercury primarily targets the cerebral hemispheres.
(C) Elemental mercury vapor primarily targets the hepatic parenchyma.
(D) Organic mercury exclusively targets the gastrointestinal mucosa.

NODIA

2019
54. Cerebellar hypoplasia in neonates is best defined in the context of toxicology as:
(A) Hypertrophy and swelling of the cerebellum from acute inflammation
(B) Deep inflammation of the cerebellar meninges and local tissues
(C) Traumatic physical damage to the cerebellum occurring during birth
(D) Cerebellar underdevelopment from fetal exposure destroying cells

2015
55. Select the correct sequence representing the progression of acute inorganic mercury poisoning in the kidney.
(A) 1. Polyuria → 2. Ischemic recovery → 3. Anuria
(B) 1. Glomerular sclerosis → 2. Tubular regeneration → 3. Vasodilation
(C) 1. Proximal tubular cell necrosis → 2. Desquamation and cast formation → 3. Oliguria/Anuria
(D) 1. Anuria → 2. Normal function → 3. Tubular necrosis

2021
56. A pregnant cat consumes fish highly contaminated with methylmercury. What is the most expected applied outcome for her kittens?
(A) Severe acute renal tubular failure at birth
(B) Congenital absence of functional immune systems
(C) Cerebellar hypoplasia and intention tremors
(D) Widespread massive coagulative hepatic

(B) Ergotism
(C) Fluorosis
(D) Ptyalism

2023
38. Which of the following pairs correctly identifies the main pathophysiological target of lead and its resulting hematological effect?
(A) Splenic parenchyma - Increased systemic erythrocyte production
(B) Heme synthesis enzymes - Basophilic stippling of erythrocytes
(C) Bone marrow precursor cells - Profound systemic neutrophilia
(D) Lymph node follicles - Severe circulating mature lymphopenia

2004
39. Laminar cortical necrosis is best defined as:
(A) Degeneration restricted to the white matter of the spinal cord
(B) Band-like necrosis of neurons within the cerebral cortex
(C) Extensive malacia and softening of the cerebellar folia
(D) Severe acute hemorrhage localized to the meningeal layers

2018
40. All of the following are common sources of lead exposure for cattle EXCEPT:
(A) Discarded vehicle batteries
(B) Old lead-based paints
(C) Freshly cut pasture grass
(D) Motor oil from contaminated machinery

2025
41. Applying the pathogenesis of lead toxicity, an effective therapeutic intervention must include a substance that:
(A) Induces cytochrome P450 hepatic metabolic pathways
(B) Stimulates the intestinal absorption of dietary calcium
(C) Functions as a reversible acetylcholinesterase inhibitor
(D) Chelates lead to form soluble excretable renal complexes

2022
42. In a dog presenting with gastrointestinal distress and unexplained seizures, the presence of nucleated red blood cells without severe anemia suggests an inference of:
(A) Lead toxicity
(B) Iron deficiency
(C) Copper toxicosis
(D) Cyanide poisoning

2021
43. Arsenic predominantly damages capillary endothelium. What is the immediate effect of this damage in the gastrointestinal tract?
(A) Hyperplasia and proliferation of mucosal goblet cells
(B) Acute hemorrhagic gastroenteritis and major fluid loss
(C) Severe atrophy of the outer muscularis intestinal layers
(D) Chronic granuloma formation in the deeper submucosa

2002
44. Inorganic arsenic exerts its primary cellular toxic mechanism by:
(A) Stimulating excessive and uncontrolled mitochondrial ATP production
(B) Binding sulfhydryl groups to inhibit cellular respiration enzymes
(C) Blockading neuromuscular nicotinic acetylcholine receptors at synapse
(D) Inducing rapid, widespread and progressive genomic DNA mutations

2019
45. Match the toxic heavy metals listed in List I with their classic pathological lesions listed in List II:
List I: (Toxic Heavy Metals)
P. Lead
Q. Arsenic
R. Mercury
S. Copper
List II: (Pathological Lesions)
1. Acute renal tubular necrosis with protein casts
2. Acute hemorrhagic gastroenteritis
3. Laminar cortical necrosis
4. Gun-metal kidneys
(A) P-2, Q-3, R-1, S-4
(B) P-4, Q-1, R-2, S-3
(C) P-3, Q-1, R-4, S-2
(D) P-3, Q-2, R-1, S-4

2001
46. What best defines the typical gross appearance of the stomach or abomasum in acute arsenic poisoning?
(A) Extreme dryness and diffuse mucosal pallor of the gastric tissue
(B) Extensive neoplastic proliferation of the gastric epithelial layer

NODIA

2001
107. Since fumonisins alter cell membrane sphingolipids, it can be inferred that they predominantly disrupt:
(A) Vascular endothelial integrity
(B) Osteoblast bone matrix mineralization
(C) Enamel formation in developing teeth
(D) Gastric acid secretion by parietal cells

2023
108. Fumonisin toxicity presents with distinct species-specific syndromes. All of the following are correct syndrome-species associations EXCEPT:
(A) Leukoencephalomalacia - Horses
(B) Pulmonary edema - Pigs
(C) Severe immunosuppression - Sheep
(D) Hepatotoxicity - Multiple species

2016
109. Gross examination of the lungs in a pig that died from acute fumonisin toxicity typically shows:
(A) Severe interlobular edema and frothy tracheal exudate
(B) Diffuse pulmonary collapse with completely dry parenchyma
(C) Multiple nodular granulomas throughout the parenchyma
(D) Extensive caseous necrosis of the major bronchial airways

2017
110. Ergotism is caused by the ingestion of ergot alkaloids produced by the fungus Claviceps purpurea, which parasitizes:
(A) Field corn and maize crops
(B) Peanuts and subterranean pods
(C) Cottonseed and related fibers
(D) Rye and other cereal grasses

2001
111. The primary cause-and-effect relationship in the pathogenesis of gangrenous ergotism involves ergot alkaloids causing:
(A) Profound peripheral vasodilation with subsequent core heat loss
(B) Sustained peripheral vasoconstriction causing ischemic necrosis
(C) Rapid intravascular destruction of circulating red blood cells
(D) Massive localized hemorrhage in the distal skeletal extremities

2001
112. Degnala disease in buffaloes and cattle is pathologically similar to ergotism and is defined clinically by:
(A) Progressive spastic paralysis of the hindlimbs
(B) Severe generalized tonic-clonic seizures
(C) Necrosis and sloughing of distal extremities
(D) Acute oliguric renal tubular failure

2008
113. Which correct pair identifies the mycotoxin mechanism and its resulting visible lesion?
(A) Fumonisin - Systemic capillary dilation
(B) Aflatoxin - Severe chronic laminitis
(C) Ochratoxin - Progressive corneal opacity
(D) Ergotamine - Ischemic dry gangrene

2003
114. If cattle are grazing fescue grass infected with the endophyte Neotyphodium coenophialum, the resulting "fescue foot" is applied toxicologically as being mechanistically identical to:
(A) Ergotism
(B) Fluorosis
(C) Aflatoxicosis
(D) Copper toxicity

2003
115. Match the mycotoxin disease in List I with its hallmark lesion in List II.
List I: (Mycotoxin disease)
P. Ergotism
Q. Zearalenone toxicosis
R. Fumonisin toxicosis (Equine)
S. Trichothecene toxicosis
List II: (Hallmark lesion)
1. Liquefactive necrosis of cerebral white matter
2. Radiomimetic mucosal necrosis
3. Dry gangrene of extremities
4. Vulvar hypertrophy and prolapse
(A) P-4, Q-3, R-2, S-1
(B) P-3, Q-4, R-1, S-2
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-3, S-2

2014
116. Based on the pathogenesis of ergotism, one can infer that the condition is exacerbated by which environmental factor?
(A) Extreme ambient heat and humidity
(B) Cold ambient temperatures
(C) Highly elevated relative humidity
(D) Extremely high barometric altitude

2014
117. Grossly, the affected extremities in chronic ergotism typically present as:
(A) Cold, anesthetic, and sharply demarcated
(B) Warm, swollen, erythematous, and painful

Downloaded from www.pdf.tube

necrosis

2024

57. From the presence of severe fibrinoid necrosis in the arterioles and acute tubular necrosis, one can infer an exposure to:
(A) Organic mercury
(B) Aflatoxin
(C) Fluorine
(D) Inorganic mercury

2012

58. All of the following are typical microscopic lesions of methylmercury toxicity in mammals EXCEPT:
(A) Severe centrilobular hepatic necrosis
(B) Loss of Purkinje cells in the cerebellum
(C) Neuronal necrosis in the cerebral cortex
(D) Spongiform changes in the nervous tissue

2022

59. Historically, a major source of mercury poisoning in livestock was:
(A) Ingestion of industrial mercury from broken thermometers
(B) Fungicide-treated seed grain inadvertently used as feed
(C) Pasture forage growing immediately adjacent to lead mines
(D) Accidental ingestion of discarded heavy copper wire rolls

2019

60. Copper toxicity in sheep is most commonly characterized as a:
(A) Sudden and acute onset of central nervous system failure
(B) Chronic storage phase followed by an acute hemolytic crisis
(C) Chronic wasting disease accompanied by skeletal deformities
(D) Primary progressive respiratory distress syndrome in lambs

2024

61. The acute hemolytic crisis in ovine copper toxicity is triggered by the sudden release of copper from the liver into the bloodstream, causing:
(A) Massive lipid peroxidation and intravascular hemolysis
(B) Direct suppression and inhibition of respiratory centers
(C) Sudden systemic failure of the blood coagulation cascade
(D) Rapid and profound suppression of active bone marrow cells

2025

62. The term "gun-metal kidneys" refers to the gross appearance of kidneys that are:
(A) Shrunken and pale from chronic interstitial fibrosis
(B) Multifocally spotted with small white cortical granulomas
(C) Dark, metallic blue-black from massive hemoglobinuria
(D) Diffusely bright yellow from severe generalized icterus

2024

63. Which of the following pairs INCORRECTLY matches a species with its relative sensitivity to copper toxicity?
(A) Sheep - Highly sensitive
(B) Cattle - Moderately tolerant
(C) Poultry - Highly tolerant
(D) Pigs - Highly sensitive

2007

64. In applying pathological diagnostics to a sheep carcass exhibiting severe generalized icterus, dark red urine, and a friable ochre liver, the pathologist should primarily suspect:
(A) Arsenic toxicosis
(B) Lead toxicosis
(C) Copper toxicosis
(D) Salt poisoning

2025

65. What is the correct chronological sequence of events in the pathogenesis of chronic copper toxicity in sheep?
1. Stressful event triggers release of copper into blood
2. Hepatic accumulation of copper over months
3. Massive intravascular hemolysis and hemoglobinuria
4. Acute tubular necrosis and death
(A) 1, 2, 3, 4
(B) 2, 1, 3, 4
(C) 2, 3, 1, 4
(D) 1, 3, 2, 4

NODIA

2024

66. Based on the pathogenesis of copper toxicosis, what can be inferred about the role of dietary molybdenum?
(A) Molybdenum increases intestinal absorption, exacerbating systemic toxicosis.
(B) Molybdenum exhibits no functional metabolic interaction with copper ions.
(C) Molybdenum directly triggers and accelerates the intravascular hemolytic crisis.

(D) Total absence of functional biliary excretion pathways

2010

28. Which statement correctly identifies an environmental factor influencing toxicity?
(A) Inherited genetic mutations that alter target receptor binding affinity
(B) Elevated ambient temperature increasing the inhalation of toxic vapors
(C) Fluctuations in body condition score and systemic lipid storage ratios
(D) Distinct sex-linked physiological and metabolic pathways of the host

2006

29. Synergism in toxicology refers to:
(A) A combined chemical response significantly greater than the sum of individual effects
(B) A combined chemical response significantly lower than the sum of individual effects
(C) A complete suppression of one agent's toxicity by another non-toxic compound
(D) A physical alteration of one toxic agent without changing its biological activity

2025

30. A neonate typically exhibits higher susceptibility to toxicosis than an adult. What toxicokinetic concept best explains this difference?
(A) Highly functional and fully developed blood-brain barrier structures
(B) Lowered rates of gastrointestinal absorption of ingested xenobiotics
(C) Immature hepatic metabolic pathways and reduced renal excretion rates
(D) Elevated physiological concentrations of systemic plasma binding proteins

2012

31. High dietary intake of a particular mineral causes competitive inhibition of absorption of a toxic heavy metal. This is an example of an effect driven by:
(A) Species-specific cellular susceptibility
(B) Primary anatomical route of exposure
(C) Synergistic environmental enhancement
(D) Toxicant antagonism by dietary factors

2009

32. From the observation that oral ingestion of venom often results in no clinical signs compared to envenomation via a bite, one can infer that:
(A) Venoms are biologically inert when administered via any physiological route.
(B) Ingested exposure enhances the systemic potency of complex animal venoms.
(C) Route of exposure dictates toxicity because of gastrointestinal breakdown.
(D) Host immunity completely neutralizes all foreign biological toxins and venoms.

2011

33. All of the following host factors generally influence an animal's susceptibility to a toxicant EXCEPT:
(A) Age
(B) Barometric pressure
(C) Nutritional status
(D) Species

2015

34. Compared to a massive single acute dose, chronic low-dose exposure to a toxicant typically results in:
(A) Identical pathological lesions and clinical signs
(B) Sudden metabolic saturation leading to rapid death
(C) Immediate failure of the gastrointestinal system
(D) Chronic organ damage driven by cumulative exposure

2013

35. Consider the Assertion (A) and Reason (R) given below and choose the correct option:
Assertion (A): Lead toxicity frequently results in laminar cortical necrosis in cattle.
Reason (R): Lead crosses the blood-brain barrier and causes capillary endothelial damage resulting in brain edema and ischemia.
(A) Both A and R are true and R is the correct explanation of A
(B) Both A and R are true but R is not the correct explanation of A
(C) A is true but R is false
(D) A is false but R is true

2002

36. Which cellular hallmark is a classic microscopic finding in the renal tubular epithelium of animals suffering from chronic lead poisoning?
(A) Intracytoplasmic lipid droplets
(B) Acid-fast cytoplasmic inclusions
(C) Intranuclear acidophilic inclusions
(D) Extensive renal calcium deposits

2022

37. What is the traditional clinical term used to denote lead poisoning in animals?
(A) Plumbism

Click Here to Buy book on Amazon

(D) Marked thinning of diaphyses with extensive hematomas

2017
77. Aflatoxins are primarily produced by species of which fungal genus?
(A) Aspergillus
(B) Fusarium
(C) Claviceps
(D) Penicillium

2011
78. Aflatoxin B1 undergoes hepatic biotransformation to form a reactive epoxide. What is the key cellular effect of this toxic metabolite?
(A) Inhibition of active acetylcholinesterase at neuronal junctions
(B) Direct physical lysis of circulating erythrocytes and platelets
(C) Binding DNA and RNA to impair protein synthesis and cell growth
(D) Severe block of voltage-gated calcium channels in myocytes

2008
79. Hepatocyte megalocytosis is defined pathologically as:
(A) Progressive increase in the total population of functional hepatocytes
(B) Diffuse cellular shrinkage and structural fragmentation of hepatocytes
(C) Fusion of adjacent hepatocytes to form multinucleated giant cells
(D) Marked hepatocyte and nuclear enlargement without cellular division

2019
80. Which of the following pairs correctly matches a specific microscopic lesion with chronic aflatoxicosis?
(A) Renal cortex - Widespread tubular calcification
(B) Brain - Severe spongiform encephalopathy lesions
(C) Liver - Biliary duct hyperplasia and fibrosis
(D) Lung - Severe acute alveolar pulmonary edema

2016
81. In an applied clinical setting, a flock of broilers fed moldy corn exhibits poor growth, increased susceptibility to secondary infections, and pale, enlarged livers. The immunosuppression is most likely due to:
(A) Fumonisin toxicity triggering major pulmonary congestion
(B) Aflatoxicosis causing cellular protein synthesis inhibition
(C) Zearalenone toxicosis inducing systemic hyperestrogenism
(D) Ergot alkaloid toxicosis causing peripheral vasoconstriction

NODIA

2023
82. From the observation of hepatic carcinomas in animals subjected to long-term feeding of spoiled groundnuts, one can infer that the toxin involved acts as a:
(A) Teratogen
(B) Radiomimetic agent
(C) Corrosive agent
(D) Mutagen and carcinogen

2023
83. All of the following are classic histological findings in the liver during chronic aflatoxicosis EXCEPT:
(A) Massive eosinophilic infiltration
(B) Marked hepatocyte megalocytosis
(C) Severe biliary duct proliferation
(D) Diffuse lipid fatty accumulation

2010
84. Which domestic species is considered the most sensitive to the toxic effects of aflatoxins?
(A) Cattle (especially young calves)
(B) Sheep (especially pregnant ewes)
(C) Poultry (especially young ducklings)
(D) Pigs (especially growing piglets)

2011
85. Consider the following statements concerning ochratoxicosis and citrinin toxicity:
I. Both are primarily nephrotoxic mycotoxins.
II. Ochratoxin A is produced by Aspergillus and Penicillium species.
III. They cause pronounced hepatic necrosis.
IV. Citrinin commonly causes perirenal edema in pigs.
Which of the above statements are correct?
(A) I, II and IV
(B) II, III and IV
(C) I, III and IV
(D) I and II only

2005
86. The primary target organ for Ochratoxin A in pigs and poultry is the:
(A) Liver
(B) Brain
(C) Heart
(D) Kidney

2008

87. The toxic effect of Ochratoxin A at the cellular level involves the inhibition of which critical enzyme?
(A) Neural acetylcholinesterase
(B) Phenylalanyl-tRNA synthetase
(C) Hepatic cytochrome P450
(D) Cellular superoxide dismutase

2005

88. Perirenal edema, often associated with citrinin and ochratoxin, is defined as:
(A) Fluid accumulation localized within the renal tubules
(B) Extensive hemorrhage and bleeding in the renal pelvis
(C) Edematous fluid accumulation around the renal capsule
(D) Inflammatory swelling and expansion of the main ureter

2022

89. A pig carcass at slaughter reveals grossly enlarged, pale kidneys with marked perirenal edema. The applied diagnostic priority should involve screening the feed for:
(A) Mycotoxins like aflatoxin
(B) Mycotoxins like fumonisin
(C) Toxic agricultural ergots
(D) Ochratoxin and citrinin

2021

90. Because Ochratoxin A extensively inhibits protein synthesis in the proximal convoluted tubules, one can infer that the urine of affected animals will likely show:
(A) Elevated levels of glucose and proteins
(B) Massive hematuria from vascular damage
(C) Exceptionally high urine specific gravity
(D) High concentrations of biliary pigments

2018

91. Which statement INCORRECTLY describes the pathology of ochratoxicosis?
(A) It causes acute proximal tubular necrosis.
(B) It causes severe laminar cortical necrosis in the brain.
(C) Chronic cases exhibit renal fibrosis and tubular atrophy.
(D) It can induce immunosuppression in poultry.

2014

92. The syndrome of "porcine nephropathy" is classically linked to the ingestion of feed contaminated with:
(A) Fusarium moniliforme mold
(B) Penicillium verrucosum
(C) Claviceps purpurea fungus
(D) Aspergillus flavus mold

2008

93. Fusariotoxicoses are diseases caused by mycotoxins produced by Fusarium species. Which two major categories of toxins are most prominent in this group?
(A) Trichothecenes and zearalenone
(B) Aflatoxins and ochratoxin A
(C) Fumonisins and ergot alkaloids
(D) Citrinin and patulin compounds

2007

94. Trichothecenes, such as T-2 toxin, are severely cytotoxic. Their radiomimetic effect primarily causes:
(A) Upregulation and proliferation of bone marrow stem cells
(B) Calcium deposition and mineralization of soft tissues
(C) Necrosis of rapidly dividing cells in active tissues
(D) Excessive hyperplasia and overgrowth of lung epithelium

2010

95. Consider the following statements regarding Fusariotoxicoses:
I. Zearalenone exhibits strong estrogenic activity.
II. Trichothecenes cause severe gastroenteritis and feed refusal.
III. Deoxynivalenol (DON) is also known as vomitoxin.
IV. Zearalenone primarily induces acute renal failure.
V. T-2 toxin lesions resemble those caused by acute radiation sickness.
Which of the above statements are INCORRECT?
(A) I and II
(B) III and V
(C) I, II and IV
(D) IV only

2001

96. Hyperestrogenism induced by Zearalenone is best defined clinically by which set of signs in swine?
(A) Anuria, oliguria, and severe perirenal edema
(B) Tumbling, muscular ataxia, and complete blindness
(C) Vulvar swelling and vaginal/rectal prolapse
(D) Bloody diarrhea and catastrophic marrow

Downloaded from www.pdf.tube

failure

2002

97. In applying knowledge of mycotoxicoses, a herd of pigs displaying sudden feed refusal and subsequent vomiting after consuming a new batch of grain should be suspected of ingesting:
(A) Zearalenone estrogenic mycotoxin
(B) Deoxynivalenol feed refusal toxin
(C) Aflatoxin M1 hepatotoxic compound
(D) Fumonisin B1 neurotoxic compound

2023

98. From the description of trichothecenes as "radiomimetic," one can infer that their pathology closely mimics that of:
(A) Persistent chronic viral infections
(B) Systemic heavy metal toxicosis cases
(C) Progressive nutritional deficiencies
(D) Direct ionizing radiation exposure

2012

99. Which of the following pathological lesions is most characteristic of acute T-2 toxin (trichothecene) toxicosis in poultry?
(A) Severe oral mucosal ulceration and necrosis
(B) Laminar cortical necrosis of the cerebrum
(C) Gun-metal kidneys with hemoglobinuria
(D) Diffuse centrilobular hepatic fibrosis

2004

100. Zearalenone exerts its effect by binding to which specific cellular structures?
(A) Nicotinic acetylcholine receptors
(B) Mitochondrial ribosomes
(C) Cytosolic estrogen receptors
(D) Voltage-gated sodium channels

2004

101. Fumonisin toxicity is caused primarily by toxins produced by which fungus?
(A) Fusarium verticillioides
(B) Aspergillus parasiticus
(C) Penicillium rubrum mold
(D) Claviceps purpurea fungi

2022

102. The underlying pathogenetic mechanism of Fumonisin B1 is the inhibition of ceramide synthase, resulting in:
(A) Profound depletion of active intracellular calcium stores
(B) Widespread systemic release of inflammatory histamine
(C) Total and complete blockade of cellular protein synthesis
(D) Altered sphingolipid pathway accumulating sphinganine

2014

103. Equine leukoencephalomalacia (ELEM) is pathologically defined as:
(A) Neoplastic proliferation of white blood cells in brain tissue
(B) Liquefactive necrosis of equine cerebral white matter tracts
(C) Acute viral inflammation of the cerebral meningeal coverings
(D) Progressive demyelination affecting the peripheral nerve fibers

2005

104. Which statement correctly identifies the primary target organ of fumonisin toxicity in pigs?
(A) The brain, resulting in leukoencephalomalacia
(B) The kidneys, causing acute renal tubular necrosis
(C) The lungs, inducing acute fatal pulmonary edema
(D) The bones, resulting in progressive osteodystrophy

NODIA

2012

105. Consider the Assertion (A) and Reason (R) given below and choose the correct option:
Assertion (A): Fumonisin exposure in pigs frequently leads to fatal pulmonary edema.
Reason (R): Fumonisin inhibits acetylcholinesterase, leading to massive bronchoconstriction and fluid exudation.
(A) Both A and R are true and R is the correct explanation of A
(B) Both A and R are true but R is not the correct explanation of A
(C) A is true but R is false
(D) A is false but R is true

2002

106. In an applied diagnostic scenario, a horse presents with sudden onset of circling, head pressing, and blindness after eating moldy corn. Necropsy reveals softened, yellowish areas in the cerebral subcortical white matter. The definitive diagnosis is:
(A) Acute lead poisoning syndrome
(B) Paralytic form of rabies
(C) Gangrenous form of ergotism
(D) Equine leukoencephalomalacia

(D) Molybdenum binds copper into insoluble complexes, preventing oral absorption.

2012

67. Microscopic examination of the liver during the chronic accumulation phase of copper toxicity often reveals:
(A) Apoptotic hepatocytes and pigment-laden swollen Kupffer cells
(B) Extensive portal tract fibrosis and prominent biliary hyperplasia
(C) Widespread coagulative necrosis throughout the hepatic lobules
(D) Complete absence of any observable pathological change in tissues

2004

68. Alkali disease and blind staggers are clinical syndromes classically associated with the toxicity of:
(A) Lead
(B) Fluorine
(C) Selenium
(D) Mercury

2004

69. High environmental intake of fluorine predominantly affects the ameloblasts and odontoblasts, resulting in:
(A) Severe dental fluorosis with mottled, soft, and rapidly wearing teeth
(B) Sudden, spontaneous loss of multiple incisors and premolar teeth
(C) Diffuse neoplastic proliferation of enamel-producing ameloblasts
(D) Progressive development of benign and malignant osteogenic tumors

2025

70. "Blind staggers" is best defined clinically as:
(A) Acute renal failure causing blindness
(B) A syndrome of wandering, ataxia, and apparent blindness in cattle grazing highly seleniferous plants
(C) The terminal stage of fluorine toxicity
(D) A congenital condition of sheep

2014

71. Which statement correctly describes the pathology of skeletal fluorosis?
(A) Bones become hyperplastic and extremely soft due to mineral loss.
(B) Fluoride deposition in bones causes exostoses and osteosclerosis.
(C) Pathological bone changes are completely restricted to cranial bones.
(D) Fluoride exposure triggers the total dissolution of bone matrices.

2012

72. A herd of cattle in a semi-arid region develops hoof deformities with separation at the coronary band and loss of tail hair. Applied toxicological knowledge suggests exposure to excessive:
(A) Copper
(B) Arsenic
(C) Selenium
(D) Lead

2021

73. All of the following are characteristic features of chronic selenium toxicity (alkali disease) EXCEPT:
(A) Alopecia (especially tail and mane)
(B) Sloughing of hooves
(C) Emaciation
(D) Acute hemorrhagic enteritis

2017

74. If an animal exhibits severe respiratory distress, "garlic breath," and acute death, it can be inferred that the exposure was most likely:
(A) Acute high-dose selenium
(B) Chronic low-dose selenium
(C) Chronic fluorine
(D) Acute fluorine

2017

75. Consider the following statements regarding selenium and fluorine toxicity:
I. Selenium can replace sulfur in amino acids causing abnormal keratins.
II. Dental fluorosis only occurs if exposure happens after permanent teeth have erupted.
III. Certain Astragalus species are known selenium accumulators.
Which of the above statements is/are INCORRECT?
(A) I and II
(B) I and III
(C) II only
(D) III only

2004

76. The gross pathological hallmark of severe skeletal fluorosis often observed during necropsy is:
(A) Complete depletion and absence of functional bone marrow
(B) Chalky white, irregular bony exostoses on long bones
(C) Intense red discoloration affecting articular cartilages

Click Here to Buy book on Flipkart

(C) Covered in diffuse and severe vesicular blisters
(D) Highly hypertrophied and deeply hyperkeratotic

2019
118. Organophosphate (OP) and carbamate insecticides primarily exert their acute toxicity by targeting which nervous system component?
(A) Voltage-gated sodium channels
(B) Postsynaptic GABA receptors
(C) Synaptic acetylcholinesterase
(D) Axonal myelin sheath membranes

2010
119. By inhibiting acetylcholinesterase, OP compounds cause the direct effect of:
(A) Depletion of synaptic acetylcholine stores and transmission failure
(B) Immediate structural destruction of the terminal nerve membrane
(C) Total blockade of active presynaptic neurotransmitter release
(D) Synaptic accumulation and overstimulation of acetylcholine

2025
120. Which definition best describes the "muscarinic signs" observed in OP poisoning?
(A) Muscle fasciculations, tremors, and profound weakness
(B) Severe generalized tonic-clonic seizures and coma
(C) SLUDGE syndrome signs including bradycardia
(D) Complete flaccid paralysis of the skeletal muscles

2004
121. Which statement INCORRECTLY differentiates between organophosphate and carbamate toxicity?
(A) Organophosphates often cause irreversible inhibition of acetylcholinesterase via aging.
(B) Carbamates require massive Phase I activation to become toxic.
(C) Carbamates cause a reversible inhibition of acetylcholinesterase.
(D) Both produce similar clinical signs of parasympathetic overstimulation.

2025
122. In an applied clinical setting, an animal presents with severe SLUDGE signs, miosis, and muscle tremors. The immediate pharmacological intervention should involve:
(A) Therapeutic administration of an acetylcholinesterase inhibitor
(B) Rapid intravenous infusion of calcium gluconate solution
(C) Prompt administration of therapeutic Vitamin K1 compound
(D) Administration of Atropine to block muscarinic receptors

2024
123. Because OP compounds cause overstimulation followed by depolarization block at the neuromuscular junction, one can infer that the ultimate cause of death is usually:
(A) Respiratory failure from paralysis and secretions
(B) Massive intracranial and cerebral hemorrhage
(C) Acute fulminant hepatic parenchymal failure
(D) Sudden onset of lethal ventricular fibrillation

2007
124. All of the following are common sources of OP or carbamate exposure in veterinary cases EXCEPT:
(A) Commercial agricultural crop sprays
(B) Veterinary flea and tick dermal dips
(C) Contaminated municipal water supplies
(D) Accidental anthelmintic overdosage

2001
125. Select the correct sequence of events in acute organophosphate poisoning.
(A) 1. OP binds acetylcholinesterase → 2. Acetylcholine accumulates → 3. Receptor overstimulation → 4. Depolarization block and paralysis
(B) 1. Acetylcholine accumulates → 2. OP binds receptors → 3. Enzyme degradation → 4. Paralysis
(C) 1. Receptor overstimulation → 2. OP inhibits acetylcholine release → 3. Paralysis → 4. Enzyme aging
(D) 1. Enzyme aging → 2. Depolarization block → 3. Acetylcholine accumulation → 4. Tremors

2025
126. A diagnostic finding that strongly supports OP toxicity in a deceased animal is:
(A) Widespread accumulation of heavy lead in the kidney
(B) Severe diffuse centrilobular necrosis in the liver
(C) Highly elevated circulating levels of methemoglobin

Downloaded from www.pdf.tube

(D) Reduced cholinesterase in blood or brain tissue

2021

127. Organochlorine insecticides, such as DDT, primarily cause toxicity by interfering with:
(A) Synaptic acetylcholinesterase enzyme activity
(B) Axonal sodium channels, causing neuroexcitation
(C) Oxidative phosphorylation pathways in mitochondria
(D) Enzymatic factors within the coagulation cascade

2003

128. The extreme lipid solubility of organochlorines causes the long-term effect of:
(A) Extremely rapid renal clearance and excretion
(B) Complete degradation by native soil microorganisms
(C) Tissue bioaccumulation and food chain biomagnification
(D) Immediate enzymatic breakdown in the ruminal environment

2010

129. Pyrethroid toxicity in cats is often characterized clinically by "pyrethroid toxicosis syndrome," which is defined by:
(A) Severe generalized internal hemorrhage and regenerative anemia
(B) Profound hypothermia, marked bradycardia, and deep coma
(C) Acute bilateral blindness and progressive appendicular lameness
(D) Salivation, ear/paw flicking, tremors, and seizures

2021

130. Which statement correctly identifies a severe post-mortem diagnostic limitation for organochlorine and pyrethroid toxicity?
(A) Absence of gross or microscopic lesions, requiring chemical assay
(B) Heavy pathognomonic gross lesions that are quickly lost to autolysis
(C) Large, persistent cellular inclusion bodies that resist standard staining
(D) Highly volatile active compounds that quickly evaporate from dead tissues

2004

131. Applying the mechanism of pyrethroids (prolonged sodium channel opening), what therapeutic approach is most logical for managing severe tremors and seizures?
(A) Immediate administration of a specific chemical antidote
(B) Administration of muscle relaxants or anticonvulsants
(C) Direct intravenous administration of sodium bicarbonate
(D) Inducing rapid emesis in a actively convulsing patient

NODIA

2007

132. From the fact that organochlorines are released from fat stores during periods of starvation, one can infer that:
(A) Heavily obese animals are completely immune to clinical toxicity
(B) Weight loss can trigger acute neurotoxicity from stored reserves
(C) General starvation enhances hepatic clearance of organochlorines
(D) Widespread fat necrosis is the primary lesion of this poisoning

2014

133. All of the following are properties of Type II pyrethroids (containing an alpha-cyano group) EXCEPT:
(A) They cause profound inhibition of acetylcholinesterase.
(B) They are generally more potent than Type I pyrethroids.
(C) They produce a syndrome characterized by severe salivation and choreoathetosis.
(D) They interfere with sodium channel gating.

2002

134. Which domestic species is notoriously most sensitive to permethrin (a pyrethroid) toxicity, usually due to inappropriate application of dog spot-on products?
(A) Horses
(B) Cattle
(C) Cats
(D) Sheep

2004

135. Arrange the sequence of pathogenesis for anticoagulant rodenticide toxicity.
1. Depletion of active Vitamin K-dependent clotting factors
2. Ingestion of the toxicant
3. Inhibition of Vitamin K epoxide reductase
4. Severe clinical hemorrhage

(A) 2, 1, 3, 4

(B) 2, 3, 1, 4
(C) 3, 2, 1, 4
(D) 2, 3, 4, 1

2017

136. Anticoagulant rodenticides cause clinical signs primarily by interfering with the synthesis of which specific clotting factors?
(A) I, II, V, X
(B) VIII, IX, XI, XII
(C) II, VII, IX, X
(D) III, IV, VII, XI

2003

137. Cholecalciferol (Vitamin D3) rodenticides cause toxicity by inducing the effect of:
(A) Severe systemic hypoglycemia and intractable seizures
(B) Hypercalcemia causing widespread tissue mineralization
(C) Profound generalized hypocalcemia and clinical tetany
(D) Complete and irreversible red bone marrow aplasia

2024

138. Zinc phosphide toxicity is defined pathologically by the release of which lethal gas in the acidic environment of the stomach?
(A) Cyanide gas
(B) Carbon monoxide
(C) Hydrogen sulfide
(D) Phosphine gas

2009

139. In a dog presenting with acute severe dyspnea, pale mucous membranes, and cavitary effusions 3 days after an unknown exposure, the applied diagnostic priority is:
(A) Anticoagulant rodenticide
(B) Zinc phosphide toxicosis
(C) Cholecalciferol exposure
(D) Strychnine toxicosis

2023

140. During a necropsy of an animal suspected of zinc phosphide poisoning, the pathologist detects a distinct "garlic" or "rotten fish" odor from the stomach contents. From this, one can infer that:
(A) The deceased animal consumed toxic heavy metal compounds
(B) The animal died of a severe septicemic bacterial infection
(C) Active phosphine gas poses a severe inhalation risk
(D) Hepatic aflatoxins are present in highly toxic concentrations

2024

141. All of the following are expected gross pathological findings in severe cholecalciferol toxicity EXCEPT:
(A) Massive uncoagulated blood in the thorax
(B) White chalky plaques on the epicardium and endocardium
(C) Gritty sensation upon sectioning the kidneys
(D) Mineralization of the gastric mucosal vessels

2022

142. The classic treatment for a patient exposed to a second-generation anticoagulant rodenticide (e.g., Brodifacoum) requires:
(A) A single therapeutic dose of Vitamin K3
(B) Rapid IV administration of calcium gluconate
(C) Prompt systemic administration of atropine
(D) Prolonged therapy with oral Vitamin K1

2018

143. Lantana camara is a notorious toxic plant that primarily affects ruminants. Its primary toxic principle acts as:
(A) A neurotoxin causing immediate paralysis
(B) A hepatotoxin causing intrahepatic cholestasis
(C) A cardiotoxin causing ventricular fibrillation
(D) A nephrotoxin causing acute tubular necrosis

2013

144. The cholestasis induced by Lantana camara prevents the excretion of phylloerythrin, which causes the secondary effect of:
(A) Acute intravascular hemolytic anemia
(B) Massive gastrointestinal tract bleeding
(C) Hepatogenous photosensitization
(D) Acute interstitial pulmonary emphysema

2021

145. Consider the following statements regarding pyrrolizidine alkaloid (PA) toxicity:
I. PA toxicity causes chronic hepatic failure.
II. PA toxicity induces hepatocyte megalocytosis.
III. PA toxicity primarily targets the renal glomerulus.
Which of the above statement(s) is/are correct?
(A) I and III
(B) II and III
(C) I only
(D) I and II

Click Here to Buy book on Amazon

(A) Massive pale areas of necrosis in the myocardium
(B) Chalky mineral deposits on the joint articular surfaces
(C) Widespread hemorrhages and luminal intestinal blood
(D) Severe generalized icterus of the mucous membranes

2024
185. Match the plant toxicant in List I with its specific resulting disease or lesion in List II.
List I: (Plant toxicant)
P. Bracken fern
Q. Oleander
R. Sorghum
S. Lantana camara
List II: (Disease or lesion)
1. Cyanide poisoning
2. Enzootic bovine hematuria
3. Cardiac arrest
4. Hepatogenous photosensitization
(A) P-3, Q-1, R-4, S-2
(B) P-2, Q-3, R-1, S-4
(C) P-1, Q-4, R-2, S-3
(D) P-2, Q-1, R-4, S-3

2021
186. Urea and other non-protein nitrogen (NPN) sources are fed to ruminants to:
(A) Provide a highly economical source of energy
(B) Directly supplement essential dietary fatty acids
(C) Act as a broad-spectrum luminal anthelmintic
(D) Allow rumen microbes to synthesize protein

2009
187. Toxicity occurs when urea is ingested in large amounts or by unadapted animals, leading to its rapid hydrolysis in the rumen to:
(A) Ammonia
(B) Nitrite
(C) Cyanide
(D) Oxalate

2011
188. Ruminal ammonia toxicosis is defined fundamentally as:
(A) A severe drop in ruminal pH leading to metabolic lactic acidosis
(B) The precipitation of toxic ammonia crystals within the renal tubules
(C) Systemic hyperammonemia exceeding hepatic urea cycle capacity
(D) Direct localized destruction of the rumen wall by urea crystals

NODIA

2021
189. In an applied scenario, a group of cattle accidentally consumes a large amount of urea fertilizer. They present with severe muscle tremors, salivation, and rapid breathing. The most appropriate immediate ruminal treatment is:
(A) Ruminal cold water and vinegar infusion to lower pH
(B) Pumping large volumes of warm water into the rumen cavity
(C) Quick oral administration of sodium bicarbonate powder
(D) Direct intravenous administration of concentrated glucose

2024
190. Because ammonia is highly alkaline, one can infer that during acute urea toxicity, the ruminal pH will:
(A) Drop significantly below 5.0
(B) Remain perfectly neutral at 7.0
(C) Fluctuate rapidly every few minutes
(D) Rise significantly, often exceeding 8.0

2010
191. All of the following are predisposing factors for urea toxicity in cattle EXCEPT:
(A) Sudden introduction of high levels of NPN to the diet
(B) Adequate adaptation to urea feeding over several weeks
(C) Feeding NPN with poor quality roughage lacking fermentable carbohydrates
(D) Consumption of water containing dissolved urea fertilizer

2018
192. Which statement INCORRECTLY describes the pathology of NPN toxicity?
(A) Diagnosis relies heavily on the demonstration of high ammonia levels in rumen fluid or blood.
(B) Animals die due to inhibition of the citric acid cycle by excess ammonia.
(C) The carcass exhibits widespread, severe pathognomonic inclusion bodies.
(D) Post-mortem autolysis is often extremely rapid.

2006
193. Gross lesions in urea toxicity are generally non-specific, but the rumen contents typically emit a strong odor of:

2007

156. In an applied emergency, a herd of cattle breaks into a field of drought-stressed Sorghum. Within minutes, several collapse with severe dyspnea and bright cherry-red mucous membranes. The specific antidote protocol must include:
(A) Intravenous infusion of methylene blue solution
(B) Repeated doses of therapeutic calcium EDTA
(C) Sodium nitrite and sodium thiosulfate therapy
(D) Therapeutic administration of Vitamin K1

2016

157. Because cyanide prevents cells from utilizing oxygen while blood remains fully oxygenated, one can infer that the venous blood in a peracute case will appear:
(A) Bright cherry red
(B) Dark, chocolate brown
(C) Black and uncoagulated
(D) Pale and watery

2002

158. Which statement INCORRECTLY characterizes nitrate/nitrite poisoning?
(A) The mucous membranes typically appear cyanotic or muddy-brown.
(B) Methylene blue is the treatment of choice to reduce methemoglobin.
(C) Toxicity is common in cattle consuming pigweed (Amaranthus) or oat hay.
(D) Post-mortem blood is characteristically bright cherry red.

2017

159. The pathological hallmark observed during necropsy of a cow that died from severe acute nitrate toxicity is:
(A) Severe acute necrosis of the rumen wall
(B) Dark, poorly coagulating chocolate-brown blood
(C) Pale, swollen, gun-metal colored kidneys
(D) Extensive gas bubble accumulation in the liver

2013

160. Ingestion of plants containing high levels of soluble oxalates, such as Halogeton or Amaranthus, primarily causes severe damage to which organ system?
(A) Central nervous system
(B) Cardiovascular system
(C) Renal system
(D) Respiratory system

2023

161. Soluble oxalates absorbed from the gastrointestinal tract combine with serum calcium, causing the dual effects of:
(A) Severe acute hypercalcemia and pathological bone fractures
(B) Massive internal hemorrhage and acute hepatic necrosis
(C) Progressive pulmonary edema and congestive heart failure
(D) Hypocalcemia and renal calcium oxalate precipitation

2011

162. Oak (Quercus species) poisoning is toxicologically defined by the presence of gallotannins which primarily cause:
(A) Acute renal tubular necrosis and gastroenteritis
(B) Chronic progressive congestive heart failure
(C) Diffuse macronodular hepatic cirrhosis
(D) Congenital cerebellar hypoplasia syndrome

2013

163. Which statement correctly identifies a microscopic hallmark of oxalate poisoning in the kidney?
(A) Prominent intranuclear viral inclusions in the glomerulus
(B) Birefringent rosette-shaped crystals in tubular lumens
(C) Massive infiltration of active eosinophils in the interstitium
(D) Complete structural absence of the glomerular Bowman's capsule

2023

164. Applying pathology to an unexplained cattle death: Necropsy reveals perirenal edema, pale swollen kidneys with petechiae, and severe mucoid hemorrhagic enteritis. The pasture contains abundant brush of the genus Quercus. The diagnosis is:
(A) Copper toxicity
(B) Oak poisoning
(C) Nitrate toxicity
(D) Ergotism

2023

165. Consider the following statements concerning nephrotoxic plants:
I. Pigweed (Amaranthus) can cause perirenal edema and toxic nephrosis.
II. Ruminants can adapt to high oxalate diets via specialized ruminal microbes.
III. Tannins in oak leaves cross-link cellular

Downloaded from www.pdf.tube

2009

146. Hepatogenous photosensitization is best defined as:
(A) Dermatitis from phylloerythrin accumulation reacting with light
(B) Primary dermal irritation caused by direct contact with plant sap
(C) A congenital metabolic defect in epidermal melanin production
(D) Severe solar sunburn secondary to grazing at high altitudes

2019

147. Applying knowledge of plant toxicology, an animal presenting with severe jaundice, photophobia, and sloughing of non-pigmented skin should be evaluated for exposure to:
(A) Bracken fern
(B) Lantana camara
(C) Sorghum species
(D) Amaranthus species

2019

148. Which correct pair matches the hepatotoxic plant with its defining pathological lesion?
(A) Lantana camara - Severe lipidosis and cholestasis
(B) Crotalaria - Megalocytosis and veno-occlusion
(C) Senecio - Acute hemorrhagic necrotizing pancreatitis
(D) Bracken fern - Diffuse non-functional biliary hyperplasia

2003

149. Since pyrrolizidine alkaloids are antimitotic agents, one can infer that affected hepatocytes will:
(A) Synthesize DNA but fail to divide, forming megalocytes
(B) Divide at extremely rapid rates, forming hepatic tumors
(C) Instantly undergo programed cell death and apoptosis
(D) Shrink dramatically and become heavily vacuolated

2015

150. All of the following are clinical or pathological features of chronic pyrrolizidine alkaloid toxicity EXCEPT:
(A) Hepatic fibrosis and cirrhosis
(B) Neurological signs (hepatic encephalopathy)
(C) Acute massive renal hemorrhage
(D) Ascites

2024

151. Which plant is a well-known source of toxic pyrrolizidine alkaloids?
(A) Nerium oleander
(B) Quercus species
(C) Sorghum bicolor
(D) Senecio jacobaea

2019

152. Nitrate poisoning in ruminants frequently occurs when animals consume heavily fertilized grasses or stressed crops. The toxicity is actually due to its conversion in the rumen to:
(A) Ammonia
(B) Nitrite
(C) Cyanide
(D) Urea

2007

153. Nitrite absorbed into the bloodstream exerts its toxic effect by:
(A) Binding competitively to mitochondrial cytochrome c oxidase
(B) Oxidizing hemoglobin to form methemoglobin
(C) Induing rapid and massive intravascular erythrocyte hemolysis
(D) Blocking the physiological release of oxygen from myoglobin

2019

154. Methemoglobinemia is defined pathologically as a condition where:
(A) Circulating hemoglobin concentration drops drastically
(B) Functional hemoglobin cannot release carbon dioxide gas
(C) Mature erythrocytes are saturated with excess free oxygen
(D) Hemoglobin is incapable of binding and transporting oxygen

2018

155. Consider the following statements concerning cyanogenic glycoside toxicity:
I. Sorghum species are a common source.
II. Toxicity is due to the release of hydrogen cyanide (HCN).
III. HCN binds to cytochrome c oxidase, blocking cellular respiration.
IV. The blood of affected animals is classically chocolate-brown.
Which of the above statement(s) is/are correct?
(A) I, II and III
(B) I, II and IV
(C) II, III and IV
(D) I, III and IV

NODIA

2010
175. Each option below consists of an Assertion (A) and a Reason (R). Select the correct choice.
Assertion (A): Ingestion of a small amount of Oleander leaves can cause sudden death in livestock.
Reason (R): Oleander contains cardiac glycosides that cause fatal cardiac arrhythmias.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2018
176. Gross pathology of an animal dying from chronic gossypol toxicity typically reveals:
(A) Shrunken, heavily fibrotic cardiac heart valves
(B) Massive focal hemorrhages within the brain tissue
(C) Dilated heart with pulmonary edema and hepatomegaly
(D) Crystalline mineral deposits in the renal pelvis

2017
177. Bracken fern (Pteridium aquilinum) contains a primary toxic principle known as ptaquiloside, which acts as:
(A) A potent and selective peripheral vasoconstrictor agent
(B) An irreversible synaptic acetylcholinesterase inhibitor
(C) A highly active cardiotoxic cardiac glycoside compound
(D) A radiomimetic bone marrow suppressor and carcinogen

2017
178. In cattle, the chronic ingestion of bracken fern causes bone marrow suppression, leading to the clinical effect of:
(A) Widespread hemorrhages due to thrombocytopenia
(B) Severe generalized absolute polycythemia state
(C) Massive leukocytosis and neutrophil overproduction
(D) Hypercoagulability and uncontrolled blood clotting

2016
179. Enzootic bovine hematuria is pathologically defined as:
(A) Acute hemoglobinuria caused by copper-induced hemolysis
(B) Bladder bleeding from ptaquiloside-induced neoplasms
(C) Hematuria secondary to severe bacterial kidney infection
(D) Pigmenturia and red urine caused by skeletal muscle breakdown

2015
180. Which statement correctly identifies the neurotoxic effect of bracken fern in horses?
(A) Direct toxicity and destruction of cerebellar Purkinje cells
(B) Thiaminase-induced thiamine deficiency and polioencephalomalacia
(C) Severe inflammatory swelling of the intraocular optic nerve
(D) Direct inhibition of acetylcholinesterase in brain tissue

2022
181. Applying the knowledge of bracken fern toxicity in horses, what is the most appropriate immediate treatment for a horse presenting with severe ataxia and tremors after grazing in a fern-rich pasture?
(A) Intravenous thiamine supplementation
(B) Intravenous calcium gluconate therapy
(C) Direct Vitamin K1 therapy administration
(D) Standard systemic atropine administration

2008
182. Based on the mechanism of ptaquiloside toxicity in cattle, one can infer that a common concurrent finding alongside hemorrhage is:
(A) Extreme systemic and arterial hypertension
(B) Rapid and uncontrolled metabolic weight gain
(C) Immunosuppression and infection from leukopenia
(D) Severely elevated circulating serum hypercalcemia

2016
183. All of the following are clinical or pathological features of bracken fern toxicity in cattle EXCEPT:
(A) Hemorrhagic diathesis
(B) Enzootic hematuria
(C) Tumors of the urinary bladder
(D) Spastic paresis of the hindlimbs

2001
184. The gross post-mortem findings of acute bracken fern toxicity in a cow are characterized by:

Click Here to Buy book on Flipkart

pneumotoxicity)
(D) Brain and spinal cord (neurotoxicity and myelotoxicity)

2017

204. Ionophore antibiotics (e.g., monensin) alter the transport of cations across cell membranes, leading to intracellular calcium overload and the direct effect of:
(A) Intense cellular proliferation of the bone marrow
(B) Severe ulcerative colitis and mucosal ulceration
(C) Massive necrosis of skeletal and cardiac muscle
(D) Rapid and progressive structural myelin degradation

2008

205. Match the veterinary drug or household chemical in List I with its hallmark pathological effect in List II.
List I: (Veterinary drug/household chemical)
P. Aminoglycosides
Q. Ionophores
R. Ethylene glycol
S. Acetaminophen (in cats)
List II: (Hallmark pathological effect)
1. Calcium oxalate crystal nephropathy
2. Myocardial necrosis
3. Acute tubular necrosis without crystals
4. Heinz body anemia and methemoglobinemia
(A) P-3, Q-2, R-1, S-4
(B) P-2, Q-3, R-1, S-4
(C) P-3, Q-1, R-4, S-2
(D) P-1, Q-4, R-2, S-3

2017

206. Ethylene glycol crystal nephropathy is defined pathologically by the accumulation of which type of crystals in the renal tubules?
(A) Microscopic urate crystals
(B) Magnesium struvite crystals
(C) Hexagonal cystine crystals
(D) Calcium oxalate crystals

2017

207. Applying toxicokinetics to an ethylene glycol poisoning case, the administration of fomepizole or ethanol is performed to:
(A) Directly dissolve existing calcium oxalate crystals in kidneys
(B) Inhibit alcohol dehydrogenase, preventing toxic metabolites
(C) Enhance systemic absorption of parent ethylene glycol in liver
(D) Induce rapid, severe osmotic diuresis within renal tubules

2011

208. Given the profound muscle damage caused by ionophore toxicity in horses, one can logically infer that serum biochemistry will show massive elevations in:
(A) Blood urea nitrogen (BUN) and creatinine
(B) Alanine aminotransferase (ALT) only
(C) Creatine kinase (CK) and cardiac troponins
(D) Bilirubin and bile acids

2021

209. All of the following species are highly susceptible to fatal monensin (ionophore) toxicity at relatively low doses EXCEPT:
(A) Horses
(B) Dogs
(C) Poultry
(D) Cattle

2002

210. Match the toxicant or drug in List I with its specific targeted antidote or treatment in List II.
List I: (Toxicant/drug)
P. Organophosphates
Q. Ethylene glycol
R. Acetaminophen
S. Anticoagulant rodenticides
List II: (Antidote or treatment)
1. Fomepizole or Ethanol
2. Vitamin K1
3. Atropine and Pralidoxime (2-PAM)
4. N-acetylcysteine
(A) P-3, Q-1, R-4, S-2
(B) P-4, Q-3, R-2, S-1
(C) P-3, Q-2, R-1, S-4
(D) P-1, Q-4, R-2, S-3

****

NODIA

(A) Bitter almonds
(B) Garlic
(C) Rotten eggs
(D) Ammonia

2009
194. Non-steroidal anti-inflammatory drugs (NSAIDs) exhibit their therapeutic and toxic effects primarily by inhibiting which enzyme?
(A) Cyclooxygenase (COX)
(B) Lipoxygenase
(C) Acetylcholinesterase
(D) Monoamine oxidase

2010
195. Each option below consists of an Assertion (A) and a Reason (R). Select the correct choice.
Assertion (A): NSAID toxicity frequently causes severe gastric ulceration.
Reason (R): NSAIDs inhibit protective prostaglandins that maintain gastric mucosal blood flow and mucus secretion.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2013
196. In the kidney, NSAID-induced inhibition of prostaglandins causes marked vasoconstriction, leading directly to the pathological effect of:
(A) Massive glomerular amyloidosis
(B) Renal papillary necrosis
(C) Proliferation of the renal pelvis epithelium
(D) Hydronephrosis

2003
197. Diclofenac toxicity in avian scavengers (vultures) is defined by its devastating ecological impact causing:
(A) Acute visceral gout and renal failure
(B) Severe hepatic lipidosis and sudden death
(C) Fragile eggshell thinning from calcium disruption
(D) Progressive neurological paralysis and starvation

2012
198. Applying pathology to a dog exhibiting melena, vomiting "coffee grounds," and severe abdominal pain after owners administered human ibuprofen. The primary lesion is likely:
(A) Severe and acute hemorrhagic cystitis
(B) Highly acute necrotizing pancreatitis
(C) Gastric erosion and deep ulceration
(D) Progressive fibrotic esophageal strictures

2023
199. From the pathogenesis of NSAID toxicity in the kidney, one can infer that toxicity is severely exacerbated by:
(A) High protein diets
(B) Concurrent use of antibiotics
(C) Cold environmental temperatures
(D) Dehydration and hypovolemia

2006
200. Each option below consists of an Assertion (A) and a Reason (R). Select the correct choice.
Assertion (A): Cats are highly sensitive to acetaminophen, rapidly developing methemoglobinemia and Heinz body anemia.
Reason (R): Cats possess high levels of glucuronyl transferase which converts acetaminophen into a highly toxic methemoglobin-forming radical.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

NODIA

2023
201. All of the following are typical clinical or pathological signs of severe NSAID toxicosis in a dog EXCEPT:
(A) Perforating gastric ulcer
(B) Severe generalized exostoses
(C) Renal crest necrosis
(D) Hypoalbuminemia due to protein-losing enteropathy

2018
202. The presence of white, chalky deposits of uric acid crystals on the serosal surfaces of organs in a vulture suggests exposure to:
(A) Lead
(B) Organophosphates
(C) Strychnine
(D) Diclofenac

2008
203. Aminoglycoside antibiotics (e.g., gentamicin) are notorious for causing toxicity to which two major organ systems?
(A) Kidneys and inner ear (nephrotoxicity and ototoxicity)
(B) Liver and spleen (hepatotoxicity and splenotoxicity)
(C) Heart and lungs (cardiotoxicity and

proteins causing necrosis.
IV. Oxalate crystals cause mechanical damage to renal epithelial cells.
V. Oak toxicity strictly spares the gastrointestinal tract.
Which of the above statement(s) is/are correct?
(A) I, II, III and IV
(B) II, III, IV and V
(C) I, III, IV and V
(D) I, II, IV and V

2009
166. From the pathogenesis of oxalate poisoning, it can be inferred that a rapid, acute manifestation of clinical signs is primarily driven by:
(A) Progressive post-renal uremia
(B) Sudden onset of liver failure
(C) Acute hypocalcemic tremors and tetany
(D) Severe intravascular hemolysis

2016
167. All of the following are true regarding Oak (Quercus) poisoning EXCEPT:
(A) Young buds and green acorns are the most toxic parts.
(B) The toxic principle is gallotannins.
(C) Gross lesions include severe ulceration of the gastrointestinal tract.
(D) It causes acute massive hepatic necrosis as its primary lesion.

2017
168. Ruminants are generally more resistant to moderate levels of oxalates than monogastrics because:
(A) They do not absorb soluble liquids from the rumen
(B) Their kidneys clear and excrete oxalates much faster
(C) Ruminal Oxalobacter formigenes degrades oxalates
(D) They possess exceptionally high systemic calcium levels

2011
169. Oleander (Nerium oleander) is an extremely toxic ornamental plant known to possess high levels of:
(A) Cyanogenic glycosides
(B) Cardiac glycosides
(C) Pyrrolizidine alkaloids
(D) Soluble oxalates

2006
170. Cardiac glycosides cause toxicity through the inhibition of the Na^+/K^+ ATPase pump, leading to the cellular effect of:
(A) Decreased intracellular calcium causing diastolic cardiac arrest
(B) Immediate massive ischemic necrosis of the working myocardium
(C) Complete blockade of conduction at the atrioventricular node
(D) Increased intracellular calcium causing severe arrhythmias

2010
171. Gossypol is a natural polyphenolic toxin best defined by its presence in the seeds of:
(A) Cotton
(B) Corn
(C) Wheat
(D) Soybeans

2023
172. Which of the following pairs INCORRECTLY associates the toxin with its primary pathological effect?
(A) Oleandrin - Severe cardiac arrhythmias and sudden death
(B) Gossypol - Congestive heart failure and myocardial degeneration
(C) Gossypol - Massive acute renal tubular necrosis
(D) Taxine (Yew) - Conduction block and sudden cardiac arrest

2005
173. In an applied agricultural scenario, feeding a large quantity of un-detoxified cottonseed meal to young pigs will most likely result in:
(A) Congestive heart failure and pulmonary edema
(B) Acute bilateral cortical blindness and deficits
(C) Severe and acute hemorrhagic gastroenteritis
(D) Dry peripheral gangrene of the distal tail

2016
174. Given that adult ruminants are relatively resistant to gossypol compared to monogastrics and pre-ruminants, one can infer that:
(A) Adult ruminants lack sensitive cardiac muscle tissues
(B) Ruminants excrete gossypol entirely through active exhalation
(C) Cottonseed is entirely unpalatable to adult grazing cattle
(D) Ruminal proteins bind free gossypol, preventing absorption

Downloaded from www.pdf.tube

ANSWERS

1. (B) Any chemical substance foreign to the biological system of the animal
Xenobiotics are chemical compounds that are entirely foreign to a living organism's normal biochemistry. These substances include synthetic drugs, environmental pollutants, food additives, and industrial chemicals. Unlike endogenous compounds, xenobiotics are not naturally produced by the host and must undergo biotransformation in the liver to facilitate their safe excretion from the body.

2. (D) The pathological condition or disease state resulting from exposure to a toxicant
Toxicosis represents the clinical and pathological disease state that manifests in an animal following exposure to a toxic substance. This term refers to the actual poisoning or biological impairment caused by the toxicant, rather than the physical substance itself. It encompasses all functional disruptions, cellular lesions, and physical symptoms associated with the toxic exposure.

3. (A) Ammonia generated from ruminal microbial degradation of urea
Endogenous toxicants are harmful substances produced within the body of an animal as a result of normal or abnormal metabolic processes. Excessive urea degradation by ruminal microbes generates toxic levels of ammonia internally, exemplifying this category. In contrast, organophosphates, mycotoxins, and heavy metals are introduced from external environmental sources and are classified as exogenous toxicants.

4. (C) Toxins are of biological origin, whereas toxicants include all poisonous substances.
Toxins are specifically defined as poisonous substances produced naturally by living organisms, such as plants, animals, or microorganisms. Toxicants is a broader term encompassing all poisonous materials, including synthetic chemicals, heavy metals, and natural toxins. Therefore, all toxins are toxicants, but synthetic chemical pollutants and industrial wastes are classified solely as toxicants rather than biological toxins.

5. (B) II and III
Phytotoxins are biological poisons produced by plants, while venoms represent a specialized class of toxins actively delivered through animal stings or bites. Xenobiotics are not exclusively man-made, as they encompass any chemical foreign to the biological system, including natural non-nutritive compounds. Thus, the classification of plant-derived toxins and animal venoms remains scientifically accurate.

6. (D) Mutagen - Substance preventing cellular DNA mutations
Mutagens are physical or chemical agents that induce permanent changes or alterations in the cellular DNA sequence, rather than preventing them. Conversely, carcinogens actively promote malignant tumor development, teratogens cause structural fetal malformations during embryonic growth, and the lethal dose fifty indicates the specific quantity of a toxic agent required to kill half of an exposed population.

NODIA

7. (B) A single exposure to a massive dose of a toxic agent
Acute poisoning typically occurs rapidly following a single, sudden exposure to a highly concentrated or massive dose of a toxic substance. This contrasts with chronic toxicity, which develops gradually over a prolonged period due to repeated, low-level exposure to a harmful compound that accumulates in tissues or causes progressive, irreversible functional damage over time.

8. (C) Fungal biological toxins
Mycotoxins are naturally occurring, secondary toxic metabolites synthesized by various species of fungi, including mold contaminants of agricultural crops. Because they are produced by living organisms, mycotoxins are categorized as biological toxins rather than synthetic agricultural chemicals, endogenous metabolites, or inorganic xenobiotics. Their ingestion leads to distinct clinical syndromes known collectively as mycotoxicoses.

9. (D) Oxidation, reduction, and hydrolysis reactions
Phase I biotransformation involves primary enzymatic modifications that introduce or expose polar functional groups on a xenobiotic. These reactions principally include oxidation, reduction, and hydrolysis, which are largely catalyzed by the hepatic cytochrome P450 enzyme system. These structural modifications prepare the lipophilic parent compound for subsequent Phase II conjugation reactions to enhance solubility.

10. (A) The movement of an absorbed toxicant via blood to tissues
Toxicokinetic distribution describes the physiological process by which an absorbed xenobiotic is transported throughout the body via systemic blood circulation to various tissues and organs. This movement depends on factors like regional blood flow, membrane permeability, tissue affinity, and plasma protein binding, which determine the concentration of the toxicant at its active site of action.

11. (B) It will require Phase II conjugation prior to renal excretion.
Lipophilic xenobiotics easily cross biological membranes but cannot be efficiently excreted by the kidneys because they undergo extensive passive reabsorption in the renal tubules. To facilitate elimination, these lipid-soluble compounds must undergo Phase II conjugation reactions. This process attaches polar molecules, increasing water solubility and allowing the metabolites to be excreted in urine or bile.

12. (B) Splenic sequestration
Xenobiotic excretion primarily occurs through active physiological pathways that remove foreign compounds from the body, such as renal filtration, biliary secretion into feces, and pulmonary exhalation of volatile substances. Splenic sequestration is a physiological process that filters and stores damaged erythrocytes or microorganisms within the spleen, playing no direct role in the elimination of chemical toxins.

13. (A) The therapeutic drug will be metabolized and excreted more rapidly.
Induction of cytochrome P450 enzymes increases the concentration and metabolic capacity of these hepatic proteins. When an animal receives a drug metabolized by this same pathway, the induced enzymes degrade the therapeutic agent at an accelerated rate. This results in lowered systemic concentrations of the drug and a significantly shorter duration of its therapeutic efficacy.

14. (C) It involves conjugation with polar molecules to increase solubility.
Phase II biotransformation represents conjugation reactions where an endogenous polar molecule, such as glucuronic acid, sulfate, or glutathione, is enzymatically attached to a xenobiotic. This step significantly increases the water solubility of the compound, rendering it highly polar. This structural modification facilitates rapid renal or biliary excretion and typically leads to detoxification of the chemical.

15. (B) II, III and IV
Enterohepatic recirculation prolongs the residence time of a toxicant rather than accelerating its elimination. Glucuronidation is a Phase II conjugation reaction, not a Phase I reaction. Additionally, the blood-brain barrier is highly permeable to lipophilic compounds, failing to exclude them. Only the assertion that gastric absorption favors weak acids is physiologically correct.

16. (C) Direct systemic circulation entry bypassing first-pass hepatic clearance
Inhaled toxic gases pass directly through the alveolar membrane into the pulmonary capillaries, entering systemic arterial circulation immediately. This rapid route bypasses the portal vein and the first-pass hepatic metabolism that occurs with orally ingested toxins. Consequently, inhalation leads to rapid distribution to target organs, causing systemic clinical signs much faster than dermal absorption.

17. (B) Liver
The liver is the primary anatomical site for xenobiotic biotransformation in animals due to its high concentration of metabolizing enzymes. It contains rich stores of cytochrome P450 mixed-function oxidases and various conjugating enzymes. Although kidneys, lungs, and intestines possess some metabolic capacity, the liver performs the vast majority of chemical alterations to detoxify or activate xenobiotics.

Click Here to Buy book on Amazon

18. (D) Antioxidant defenses
Oxidative stress occurs when the cellular production of highly reactive oxygen species exceeds the neutralization capacity of endogenous antioxidant defenses. These defensive systems include enzymes like superoxide dismutase, catalase, and glutathione peroxidase, as well as non-enzymatic molecules. When these protective mechanisms are overwhelmed, reactive oxygen species damage cellular lipids, proteins, and DNA, leading to cell injury.

19. (A) Caspase-mediated programmed cell death with cellular fragmentation
Apoptosis is a highly regulated, energy-dependent pathway of programmed cell death mediated by caspases. It is characterized morphologically by cell shrinkage, chromatin condensation, and the formation of apoptotic bodies with intact plasma membranes. Unlike necrosis, this physiological process prevents the leakage of cellular contents, thereby avoiding the recruitment of inflammatory cells and sparing surrounding tissues.

20. (C) Dysfunction of Na^+/K^+ ATPase pumps resulting in cellular swelling
Depletion of cellular ATP impairs the active sodium-potassium ATPase pump located in the plasma membrane. This failure leads to intracellular accumulation of sodium and loss of potassium, creating an osmotic gradient that drives water into the cell. Consequently, acute ATP depletion manifests morphologically as rapid cellular swelling, organelle dilation, and hydropic degeneration.

21. (A) Intact cell membranes
Necrotic cell death is characterized by the loss of plasma membrane integrity, leading to the leakage of intracellular enzymes and inflammatory responses. Microscopic hallmarks of necrosis include increased cytoplasmic eosinophilia, nuclear changes such as karyolysis, pyknosis, or karyorrhexis, and localized inflammation. In contrast, maintaining intact cell membranes during cellular breakdown is a defining feature of apoptotic cell death.

22. (D) Rapid failure of oxidative phosphorylation and ATP synthesis
Disrupting the mitochondrial inner membrane abolishes the electrochemical proton gradient necessary to drive the F1F0-ATPase enzyme. This disruption halts the process of oxidative phosphorylation, leading to a catastrophic drop in cellular ATP synthesis. Cells deprived of ATP rapidly experience homeostatic failure, membrane pump dysfunction, swelling, and subsequent acute necrotic or apoptotic death.

NODIA

23. (B) Membrane damage exclusively affects the plasma membrane, sparing organelles.
Membrane damage during toxic cell injury is a widespread process that affects both the outer plasma membrane and intracellular organelle membranes. Free radicals damage mitochondrial membranes, leading to cytochrome c release, and disrupt lysosomal membranes, releasing hydrolytic enzymes into the cytoplasm. This organellar destruction occurs alongside plasma membrane damage, accelerating cell death through multiple pathways.

24. (C) Severe acute toxic insult often triggers overlapping features of both death pathways.
Distinguishing between apoptosis and necrosis purely on morphological criteria is difficult because severe acute toxic insults can damage cells to varying degrees simultaneously. A single toxicant can trigger apoptosis at low concentrations but cause widespread necrosis at higher doses. This concentration gradient results in overlapping cellular features, making precise morphological separation challenging in tissue specimens.

25. (B) I, II, IV and V
Calcium influx activates intracellular enzymes like phospholipases, while mitochondrial cytochrome c release initiates the apoptotic caspase cascade. Lipid peroxidation yields reactive aldehydes, and glutathione acts as a crucial cellular antioxidant. Superoxide dismutase acts as a protective scavenger that detoxifies free radicals rather than generating them, making the other four pathogenetic statements correct.

26. (A) An iron chelator or antioxidant
Lipid peroxidation is driven by free radical chain reactions that damage hepatocyte membranes, a process catalyzed by free intracellular iron through the Fenton reaction. Administering an iron chelator minimizes iron availability, while antioxidants directly scavenge propagating free radicals. This combined therapeutic approach halts the peroxidation cascade, preserving membrane integrity and preventing further cellular necrosis.

tolerance. Classifying pigs as highly sensitive to copper is therefore incorrect in veterinary toxicology.

64. (C) Copper toxicosis
A sheep carcass presenting with generalized icterus, dark red-to-black urine, and a pale yellow, friable liver is highly characteristic of acute copper toxicosis. These lesions reflect the catastrophic intravascular hemolysis and subsequent hemoglobinuria that define the terminal phase of chronic copper poisoning. This specific clinical picture warrants immediate diagnostic confirmation via tissue copper analysis.

65. (B) 2, 1, 3, 4
Chronic copper toxicity begins with the silent accumulation of copper in hepatocytes over several months. A subsequent stressful event triggers the sudden, massive release of this stored copper into the bloodstream. This free copper causes extensive intravascular hemolysis and hemoglobinuria. Finally, the high load of circulating hemoglobin induces acute renal tubular necrosis, leading to death.

66. (D) Molybdenum binds copper into insoluble complexes, preventing oral absorption.
Dietary molybdenum interacts directly with copper and sulfur in the rumen to form insoluble copper thiomolybdates. This biochemical interaction prevents the absorption of copper from the gastrointestinal tract, promoting its excretion in the feces. Consequently, adequate dietary molybdenum levels protect sheep from copper accumulation, while low molybdenum levels increase the risk of chronic copper toxicosis.

67. (A) Apoptotic hepatocytes and pigment-laden swollen Kupffer cells
During the subclinical storage phase of copper toxicity, microscopic evaluation of the liver reveals individual hepatocyte degeneration, increased apoptosis, and swollen Kupffer cells containing copper-rich pigment. These subtle, progressive cellular changes occur before the catastrophic release of copper into the bloodstream. This ongoing damage eventually compromises hepatic storage, culminating in the acute hemolytic crisis.

68. (C) Selenium
Alkali disease and blind staggers are distinct clinical syndromes historically and pathologically associated with selenium poisoning in livestock. These conditions develop when animals graze on soils rich in selenium or consume selenium-accumulating plants. Alkali disease represents the chronic form, while blind staggers was traditionally associated with subacute exposure to specific seleniferous plant species in semi-arid ranges.

69. (A) Severe dental fluorosis with mottled, soft, and rapidly wearing teeth
High fluoride intake during tooth development damages ameloblasts and odontoblasts, impairing enamel and dentin formation. This damage results in dental fluorosis, characterized by mottled, stained, soft, and structurally weak teeth that wear down rapidly. This lesion only develops if exposure occurs before the permanent teeth erupt, leading to painful chewing and subsequent emaciation in livestock.

70. (B) A syndrome of wandering, ataxia, and apparent blindness in cattle grazing highly seleniferous plants
Blind staggers is a clinical syndrome traditionally observed in cattle grazing on highly seleniferous vegetation. Affected animals display characteristic neurological signs, including wandering, head pressing, ataxia, circling, and apparent blindness. While historical accounts linked this directly to selenium, modern research indicates that sulfur compounds and other plant toxins may also contribute to this complex pasture syndrome.

71. (B) Fluoride deposition in bones causes exostoses and osteosclerosis.
Chronic fluoride ingestion alters normal bone remodeling, replacing normal mineral structures with abnormal, dense bone tissue. This pathological process leads to osteosclerosis and the formation of painful bony exostoses, especially on the metatarsals, metacarpals, and ribs. These skeletal changes cause progressive lameness, joint stiffness, and physical discomfort, severely affecting the mobility and productivity of animals.

72. (C) Selenium
Hoof deformities, separation of the hoof wall at the coronary band, and progressive loss of long

Downloaded from www.pdf.tube

36. (C) Intranuclear acidophilic inclusions
Chronic lead poisoning characteristically produces distinct intranuclear acidophilic inclusion bodies within the epithelial cells of the proximal convoluted tubules. These inclusions consist of a lead-protein complex and are highly diagnostic when visualized microscopically. Their presence represents a cellular mechanism to sequester free lead ions, protecting organelle function from the toxic effects of the heavy metal.

37. (A) Plumbism
Plumbism is the historical and clinical term used in medicine and veterinary pathology to specifically denote lead poisoning. This designation originates from the Latin word for lead, plumbum. Identifying this condition is crucial in veterinary diagnostics, as lead exposure remains a common cause of acute neurological and gastrointestinal disease in cattle, dogs, and waterfowl.

38. (B) Heme synthesis enzymes - Basophilic stippling of erythrocytes
Lead directly inhibits critical enzymes in the heme synthesis pathway, particularly delta-aminolevulinic acid dehydratase and ferrochelatase. This inhibition prevents proper iron incorporation into heme and causes accumulation of ribosomal RNA degradation products in red blood cells. Microscopically, this manifests as basophilic stippling of erythrocytes, a highly characteristic hematological finding in lead toxicity.

39. (B) Band-like necrosis of neurons within the cerebral cortex
Laminar cortical necrosis is characterized pathologically by a distinct, band-like pattern of neuronal degeneration and necrosis restricted to specific middle layers of the cerebral cortex. This lesion often results from severe cerebral ischemia, hypoxia, or metabolic disruption. Under ultraviolet light, the necrotic bands of the cerebral cortex frequently exhibit auto-fluorescence, aiding in diagnosis.

40. (C) Freshly cut pasture grass
Cattle are frequently exposed to lead through discarded vehicle batteries, old lead-based paints on barns, and contaminated used motor oil. These sources contain high concentrations of bioavailable lead. Freshly cut pasture grass is a clean, natural forage source and is not a common vehicle for lead exposure, unless the pasture soil itself has severe industrial contamination.

41. (D) Chelates lead to form soluble excretable renal complexes
Effective treatment for lead poisoning requires administering a chelating agent, such as calcium disodium EDTA. This compound binds circulating and tissue-bound lead ions, forming stable, water-soluble complexes. These chelated complexes are non-toxic and can be easily excreted from the body through renal filtration, preventing further cellular damage in target tissues like the brain and kidneys.

NODIA

42. (A) Lead toxicity
In dogs, lead poisoning characteristically disrupts erythrocyte maturation without necessarily causing severe anemia initially. This disruption leads to the premature release of nucleated red blood cells into systemic circulation. When this unusual hematological finding is accompanied by gastrointestinal distress and acute neurological signs like seizures, lead toxicity is a primary differential diagnosis that warrants immediate testing.

43. (B) Acute hemorrhagic gastroenteritis and major fluid loss
Arsenic damages capillary endothelial cells, leading to widespread vascular dilatation, increased permeability, and subsequent thrombosis. In the gastrointestinal tract, this capillary injury causes extensive mucosal ischemia, necrosis, and sloughing. The clinical result is severe hemorrhagic gastroenteritis, characterized by profuse watery diarrhea, massive blood loss, dehydration, and rapid cardiovascular shock in affected animals.

44. (B) Binding sulfhydryl groups to inhibit cellular respiration enzymes
Inorganic arsenic binds tightly to sulfhydryl groups on critical cellular enzymes, particularly those in the pyruvate dehydrogenase complex. This binding halts the citric acid cycle and impairs cellular respiration, preventing ATP production. Tissues with high metabolic rates, such as the intestinal mucosa and capillary endothelium, are highly sensitive to this energy failure, leading to acute necrosis.

45. (D) P-3, Q-2, R-1, S-4
Lead poisoning characteristically causes laminar cortical necrosis in the brain of ruminants. Arsenic causes acute hemorrhagic gastroenteritis due to capillary endothelial damage. Inorganic mercury targets the kidneys, causing acute renal tubular necrosis with protein casts. Copper toxicosis triggers a massive hemolytic crisis in sheep, leading to highly characteristic hemoglobinuric gun-metal kidneys.

46. (C) Severe mucosal edema with intense "rose-red" hyperemic inflammation
Acute arsenic poisoning causes a characteristic rose-red hyperemic inflammation of the gastric or abomasal mucosa. This intense discoloration is accompanied by severe submucosal edema, mucosal necrosis, and sloughing. The lesion results from direct capillary damage and vasodilation within the gastrointestinal wall, providing a highly suggestive post-mortem finding during necropsy of affected livestock.

47. (D) It acts as a pure neurotoxin sparing the gastrointestinal tract.
Arsenic is not a pure neurotoxin, as its primary pathophysiological target is the vascular endothelium, causing severe damage in the gastrointestinal tract. While neurological signs can occur due to systemic shock or direct nerve damage, the hallmark of arsenic poisoning is acute, severe hemorrhagic gastroenteritis. It is commonly found in herbicides, pesticides, and wood preservatives.

48. (C) Arsenic toxicity
In sheep, the sudden onset of profuse, watery, bloody diarrhea accompanied by intense abomasal hyperemia on post-mortem is highly suggestive of acute arsenic poisoning. This heavy metal directly targets and destroys the capillary beds of the gastrointestinal mucosa, leading to severe fluid loss and hemorrhage. These findings warrant prioritizing diagnostic testing for arsenic in tissue and feed.

49. (B) Arsenic
Severe paralysis of capillary beds, accompanied by increased vascular permeability and plasma exudation, is the classic pathophysiological hallmark of arsenic poisoning. Arsenic directly targets endothelial cells, leading to profound vasodilation, fluid leakage, and subsequent tissue ischemia. This vascular damage is most pronounced in the gastrointestinal tract, resulting in characteristic edema, congestion, and hemorrhagic lesions.

50. (D) Anthelmintic and growth promoter
Historically, arsenic compounds were widely utilized in veterinary medicine as anthelmintics, tonic preparations, and growth promoters in poultry and swine feed. Before the development of modern therapeutics, these compounds were common agricultural additives. This historical usage frequently led to accidental poisonings when livestock gained access to concentrated farm stocks or improperly formulated feed.

51. (A) Organic methylmercury
Organic methylmercury is highly lipophilic and readily crosses the blood-brain barrier via active transport systems. Once inside the central nervous system, it accumulates and causes severe, diffuse neurotoxicity by generating oxidative stress and disrupting protein synthesis. This organic form is far more neurotoxic than elemental or inorganic mercury salts, which do not cross this barrier efficiently.

52. (C) Proximal convoluted tubules
Inorganic mercury salts are primarily concentrated and accumulated within the epithelial cells of the renal proximal convoluted tubules. The kidneys filter and actively transport these heavy metal ions, leading to intracellular oxidative stress and enzyme inhibition. This accumulation results in severe, acute renal tubular necrosis, presenting clinically as oliguric or anuric kidney failure in affected animals.

53. (A) Methylmercury primarily targets the central nervous system.
Organic methylmercury is a highly lipophilic compound that selectively targets the central nervous system, readily crossing the blood-brain barrier to cause widespread neuronal damage. In contrast, inorganic mercury salts cannot easily cross this barrier and primarily target the renal proximal tubules, causing acute kidney failure. Elemental vapor targets both the lungs and the nervous system.

54. (D) Cerebellar underdevelopment from fetal exposure destroying cells
Cerebellar hypoplasia is a developmental defect characterized by the underdevelopment and reduced size of the cerebellum. In toxicology, this

Click Here to Buy book on Flipkart

occurs when a fetus is exposed in utero to agents like methylmercury, which cross the placenta and destroy rapidly dividing external germinal layer cells in the developing cerebellum, halting normal tissue maturation and causing motor deficits at birth.

55. (C) 1. Proximal tubular cell necrosis → 2. Desquamation and cast formation → 3. Oliguria/ Anuria
Ingesting inorganic mercury triggers rapid necrosis of the proximal tubular epithelial cells, which then desquamate into the tubular lumen. These sloughed cells form cellular and proteinaceous casts that physically obstruct the renal tubules. This obstruction, combined with altered vascular hemodynamics, decreases the glomerular filtration rate, resulting in clinical oliguria or complete anuria.

56. (C) Cerebellar hypoplasia and intention tremors
Methylmercury readily crosses the placental barrier in pregnant cats, targeting the developing fetal brain. The toxin selectively destroys the rapidly dividing cells of the external germinal layer in the fetal cerebellum. Consequently, kittens are born with cerebellar hypoplasia, presenting clinically with severe intention tremors, ataxia, hypermetria, and general loss of fine motor coordination.

57. (D) Inorganic mercury
Widespread fibrinoid necrosis of arterioles combined with acute proximal renal tubular necrosis is a classic pathological signature of inorganic mercury poisoning. Inorganic mercury salts directly damage vascular endothelial cells and accumulate selectively in the kidneys, causing severe tissue ischemia and tubular destruction. These structural lesions differentiate inorganic mercury exposure from other heavy metal or organic toxicities.

58. (A) Severe centrilobular hepatic necrosis
Methylmercury is primarily a neurotoxic compound that targets the central nervous system, causing cerebral neuronal necrosis, loss of cerebellar Purkinje cells, and spongiform changes in brain tissue. It does not cause severe centrilobular hepatic necrosis as a primary lesion, which is a hallmark of hepatotoxins like aflatoxins or blue-green algae rather than organic mercury.

59. (B) Fungicide-treated seed grain inadvertently used as feed
Historically, a major source of mercury poisoning in livestock was the accidental feeding of leftover seed grain treated with organomercurial fungicides. Farmers sometimes used this treated grain as animal feed instead of discarding it. This practice led to widespread poisoning, characterized by severe neurological disease from organic mercury or renal damage from inorganic metabolites.

60. (B) Chronic storage phase followed by an acute hemolytic crisis
Copper toxicity in sheep is a classic cumulative poisoning characterized by a prolonged, subclinical storage phase in hepatocytes. During this phase, copper levels build up silently without causing clinical symptoms. Once hepatic storage capacity is saturated or a stressful event occurs, copper is rapidly released into the blood, triggering a sudden, highly fatal hemolytic crisis.

61. (A) Massive lipid peroxidation and intravascular hemolysis
The sudden release of accumulated copper from the liver into the bloodstream triggers massive lipid peroxidation of erythrocyte membranes. Copper acts as a potent oxidant, damaging red blood cells and leading to rapid, widespread intravascular hemolysis. This acute breakdown of red blood cells causes severe anemia, icterus, and hemoglobinuria, which are classic clinical features of the crisis.

62. (C) Dark, metallic blue-black from massive hemoglobinuria
The term “gun-metal kidneys” describes the gross appearance of kidneys during an acute copper hemolytic crisis. The massive release of hemoglobin from ruptured red blood cells overloads the renal filtration system, leading to extensive hemoglobin casts within the tubules. This high concentration of oxidized hemoglobin imparts a characteristic dark, metallic, blue-black discoloration to the kidneys.

63. (D) Pigs - Highly sensitive
Pigs are highly tolerant of dietary copper and can consume elevated levels without adverse effects, often receiving copper as a growth promoter. Conversely, sheep are extremely sensitive to copper accumulation and toxicity. Cattle are moderately tolerant, and poultry exhibit high

Downloaded from www.pdf.tube

aflatoxin epoxides to cellular nucleic acids, which directly inhibits protein synthesis. This cellular block reduces the production of essential immunoglobulins, cytokines, and clotting factors, leaving birds highly vulnerable to secondary pathogens.

82. (D) Mutagen and carcinogen
The development of hepatic carcinomas in animals consuming aflatoxin-contaminated groundnuts demonstrates that the toxin acts as a potent mutagen and carcinogen. Aflatoxin-8,9-epoxide binds to DNA, inducing specific mutations in tumor suppressor genes. This genetic damage initiates neoplastic transformation, leading to hepatocellular carcinoma, making aflatoxin one of the most potent naturally occurring carcinogens known.

83. (A) Massive eosinophilic infiltration
Chronic aflatoxicosis is microscopically characterized by biliary duct hyperplasia, hepatocyte megalocytosis, portal fibrosis, and diffuse lipid accumulation within hepatocytes. These changes represent the chronic degenerative and regenerative hepatic response to the mycotoxin. Massive eosinophilic infiltration is a characteristic of allergic or parasitic responses and is not a typical histological feature of chronic aflatoxin-induced liver damage.

84. (C) Poultry (especially young ducklings)
While all domestic animals are susceptible to aflatoxicosis, poultry, particularly young ducklings, are considered the most sensitive species to its toxic effects. Ducklings are highly susceptible to acute liver damage and rapid biliary proliferation, making them the classic bioassay model for detecting aflatoxins. Young turkeys and broilers also exhibit high sensitivity compared to mature ruminants.

85. (A) I, II and IV
Ochratoxin A and citrinin are both major nephrotoxic mycotoxins that cause porcine nephropathy and perirenal edema. Ochratoxin A is synthesized by species of both Aspergillus and Penicillium. These toxins selectively target the kidneys rather than causing primary hepatic necrosis. These clinical and mycological characteristics confirm that three of the four assertions are correct.

86. (D) Kidney
The kidney is the primary target organ for Ochratoxin A in pigs and poultry. This mycotoxin accumulates within the renal cortex, particularly targeting the epithelial cells of the proximal convoluted tubules. Chronic ingestion leads to progressive renal degeneration, tubular atrophy, and interstitial fibrosis, a disease syndrome historically referred to as mycotoxic porcine nephropathy.

87. (B) Phenylalanyl-tRNA synthetase
Ochratoxin A exerts its primary cellular toxicity by competitively inhibiting the enzyme phenylalanyl-tRNA synthetase. This inhibition blocks the attachment of phenylalanine to its tRNA molecule, halting protein synthesis within target cells. The highly active transport systems in the renal proximal tubules concentrate the toxin, making kidney cells exceptionally vulnerable to this metabolic arrest and subsequent necrosis.

88. (C) Edematous fluid accumulation around the renal capsule
Perirenal edema is pathologically defined as the accumulation of clear, serous, or gelatinous edematous fluid within the connective tissues immediately surrounding the renal capsule. This lesion is a classic post-mortem finding in pigs exposed to nephrotoxic agents like ochratoxin, citrinin, or certain toxic weeds, reflecting severe disruption of renal vascular integrity and fluid balance.

89. (D) Ochratoxin and citrinin
The discovery of pale, enlarged kidneys accompanied by marked perirenal edema in a pig carcass is a classic diagnostic indicator of nephrotoxic mycotoxicoses. This specific pathological presentation strongly implicates exposure to Ochratoxin A and citrinin. Consequently, diagnostic protocols must prioritize testing feed samples for these specific fungal toxins to prevent further herd outbreaks.

90. (A) Elevated levels of glucose and proteins
Because Ochratoxin A damages the proximal convoluted tubule epithelium and inhibits protein synthesis, the kidneys lose their capacity to reabsorb filtered solutes. This tubular dysfunction leads to the excretion of undigested molecules in the urine. Affected animals consequently exhibit glycosuria and proteinuria, along with a marked

NODIA

tail and mane hair are classic diagnostic signs of chronic selenium toxicosis, also known as alkali disease. Selenium replaces sulfur in keratin molecules, disrupting the structural integrity of hooves and hair. This protein alteration leads to severe lameness and alopecia in affected cattle.

73. (D) Acute hemorrhagic enteritis
Chronic selenium toxicity, or alkali disease, is characterized by progressive weight loss, alopecia of the tail and mane, and severe hoof deformities with sloughing. These lesions reflect the chronic disruption of keratin synthesis. Acute hemorrhagic enteritis is a feature of acute, high-dose selenium poisoning or other corrosive toxicants, rather than the slow, cumulative syndrome of chronic selenosis.

74. (A) Acute high-dose selenium
Acute exposure to high doses of selenium causes severe pulmonary congestion, edema, and rapid cardiovascular collapse. A highly characteristic diagnostic finding in these acute cases is a distinct "garlic breath" odor, which results from the pulmonary exhalation of volatile dimethyl selenide metabolites. This clinical presentation differs significantly from the dental and skeletal lesions of chronic fluorosis.

75. (C) II only
Dental fluorosis only occurs when animals are exposed to high fluoride levels during the active development and mineralization of permanent teeth, before they erupt. Exposure after eruption does not cause developmental enamel mottling. Selenium does replace sulfur in structural proteins, and certain Astragalus species actively accumulate selenium, making the second statement the only incorrect assertion.

76. (B) Chalky white, irregular bony exostoses on long bones
During necropsy of animals with chronic fluorosis, the skeletal lesions are highly visible. Affected bones appear enlarged, heavy, and chalky white, losing their normal smooth, glistening periosteal surface. Widespread, irregular bony exostoses are frequently found along the shafts of the long bones and mandible, representing the pathological bone remodeling and mineralization typical of chronic fluorine poisoning.

77. (A) Aspergillus
Aflatoxins are a group of highly toxic mycotoxins produced primarily by soil-borne fungi belonging to the genus Aspergillus, most notably Aspergillus flavus and Aspergillus parasiticus. These molds grow readily on agricultural commodities like corn, peanuts, and cottonseed under warm, humid conditions. Ingestion of these contaminated feeds leads to severe hepatic damage and systemic disease in livestock.

78. (C) Binding DNA and RNA to impair protein synthesis and cell growth
Hepatic biotransformation of aflatoxin B1 produces a highly reactive intermediate known as aflatoxin-8,9-epoxide. This electrophilic metabolite binds covalently to cellular macromolecules, particularly DNA and RNA, forming stable adducts. This binding disrupts transcription and translation processes, leading to the inhibition of protein synthesis, impaired cell division, and subsequent hepatocyte necrosis or neoplastic transformation.

79. (D) Marked hepatocyte and nuclear enlargement without cellular division
Hepatocyte megalocytosis is a classic histopathological lesion characterized by a marked increase in both cell and nuclear size of hepatocytes without associated cellular division. This occurs because the toxicant inhibits DNA replication and mitosis while allowing cytoplasmic protein synthesis to continue. This nuclear and cellular gigantism is highly characteristic of chronic pyrrolizidine alkaloid and aflatoxin poisonings.

80. (C) Liver - Biliary duct hyperplasia and fibrosis
The liver is the primary target organ for aflatoxins, and chronic exposure produces a highly characteristic triad of microscopic lesions. These include marked biliary duct hyperplasia, bridging portal fibrosis, and hepatocyte megalocytosis. This proliferative ductular response and extensive scarring reflect the chronic regenerative attempts of the liver in response to persistent toxic hepatocyte necrosis.

81. (B) Aflatoxicosis causing cellular protein synthesis inhibition
Broilers fed aflatoxin-contaminated feed exhibit poor growth, hepatomegaly, and marked immunosuppression. This suppression of the immune system is driven by the binding of

Click Here to Buy book on Amazon

27. (B) Insufficient level of glucuronyl transferase activity
Felines have a well-documented genetic deficiency in glucuronyl transferase enzymes, limiting their capacity to perform glucuronidation. Because this Phase II conjugation pathway is highly restricted, cats cannot efficiently metabolize phenolic compounds, including acetaminophen. This deficiency leads to the accumulation of highly toxic intermediates, causing oxidative damage, Heinz body hemolytic anemia, and hepatic necrosis.

28. (B) Elevated ambient temperature increasing the inhalation of toxic vapors
Environmental factors influencing toxicosis are external conditions that affect toxicant exposure or biological activity. Elevated ambient temperatures increase the volatility and evaporation of certain chemical solvents, leading to higher concentrations of toxic vapors in the breathing zone. Genetic variations, body condition, and sex represent host-specific factors rather than environmental parameters that influence toxicity.

29. (A) A combined chemical response significantly greater than the sum of individual effects
Synergism occurs when the simultaneous exposure to two or more toxicants results in a combined biological effect that is significantly greater than the mathematical sum of their individual toxicities. This interaction dramatically amplifies toxic potential, posing severe clinical risks compared to separate exposures. It differs from additive effects or antagonistic interactions that decrease overall toxicity.

30. (C) Immature hepatic metabolic pathways and reduced renal excretion rates
Neonatal animals possess immature hepatic enzyme systems, particularly Phase II conjugating pathways, and have lower glomerular filtration rates. These physiological limitations significantly decrease their capacity to metabolize and exrate absorbed xenobiotics. Consequently, toxicants persist longer in the systemic circulation of neonates at higher concentrations, markedly increasing their susceptibility to severe poisoning.

31. (D) Toxicant antagonism by dietary factors
High dietary intake of specific essential minerals can competitively inhibit the intestinal absorption of toxic heavy metals by sharing the same transport proteins. This interaction represents a classic example of toxicant antagonism driven by dietary composition. By reducing the bioavailability of the toxicant, diet directly mitigates systemic absorption and subsequent clinical severity of the poisoning.

32. (C) Route of exposure dictates toxicity because of gastrointestinal breakdown.
Complex animal venoms consist primarily of high molecular weight proteins and polypeptides. When ingested orally, these proteins are digested and inactivated by proteolytic enzymes and acidic conditions in the gastrointestinal tract. Consequently, oral exposure rarely causes systemic toxicity, demonstrating how the primary anatomical route of exposure determines the clinical outcome of toxin contact.

33. (B) Barometric pressure
Susceptibility to toxicants is heavily influenced by internal host factors such as age, nutritional status, and species-specific metabolic differences. These physiological variables dictate how a compound is absorbed, metabolized, and excreted. Barometric pressure is an external physical environmental condition that does not generally affect the biological susceptibility of an animal to chemical poisoning.

NODIA

34. (D) Chronic organ damage driven by cumulative exposure
Chronic exposure to low doses of a toxicant allows the compound or its subclinical damage to accumulate gradually over time. This slow progression results in persistent degenerative changes, tissue fibrosis, and functional organ failure. Unlike acute high-dose exposure, which causes sudden death or rapid systemic shock, chronic poisoning manifests as a slow, progressive decline in health.

35. (A) Both A and R are true and R is the correct explanation of A
Lead poisoning in ruminants causes brain edema and ischemia by damaging capillary endothelial cells, disrupting the blood-brain barrier. This localized vascular damage and subsequent tissue swelling lead to severe hypoxia in the cerebral cortex. This lack of oxygen results in the classic pathological lesion known as laminar cortical necrosis, confirming both statements are true and causally linked.

decrease in urine concentrating ability, indicating severe renal tubular impairment.

91. (B) It causes severe laminar cortical necrosis in the brain.
Ochratoxicosis is primarily a nephrotoxic syndrome characterized by acute proximal tubular necrosis, renal fibrosis, tubular atrophy, and systemic immunosuppression. It does not cause laminar cortical necrosis in the brain, which is a lesion associated with lead poisoning, sulfur toxicity, or thiamine deficiency. Thus, describing brain necrosis as a feature of ochratoxicosis is pathologically incorrect.

92. (B) Penicillium verrucosum
Porcine nephropathy is a well-recognized chronic kidney disease in swine that is classically linked to the ingestion of feed contaminated with Penicillium verrucosum, a major producer of Ochratoxin A in temperate climates. This mold thrives on stored cereal grains, producing the toxin that causes progressive renal proximal tubular degeneration, interstitial fibrosis, and subsequent organ failure.

93. (A) Trichothecenes and zearalenone
Fusariotoxicoses encompass various clinical disease syndromes caused by mycotoxins from the fungal genus Fusarium, which produces several structurally diverse toxic compounds. The two most prominent and economically significant categories in livestock are cytotoxic trichothecenes, which cause severe gastroenteritis and tissue necrosis, and zearalenone, a non-steroidal estrogenic mycotoxin that induces severe reproductive disorders in swine.

94. (C) Necrosis of rapidly dividing cells in active tissues
Trichothecenes like T-2 toxin exert a radiomimetic effect, meaning their pathological impact mimics that of ionizing radiation. They selectively target and cause necrosis in rapidly dividing cell populations, such as those in the bone marrow, lymphoid tissues, and intestinal mucosa. This cellular destruction leads to leukopenia, anemia, immune suppression, and severe gastrointestinal hemorrhage in affected animals.

95. (D) IV only
Zearalenone is a potent estrogenic mycotoxin that targets the reproductive system, causing vulvovaginitis and reproductive failure in swine, rather than inducing acute renal failure. Conversely, trichothecenes cause severe gastroenteritis, deoxynivalenol is vomitoxin, and T-2 toxin produces radiomimetic lesions. Therefore, the statement claiming zearalenone primarily causes renal failure is the only incorrect assertion.

96. (C) Vulvar swelling and vaginal/rectal prolapse
Hyperestrogenism induced by zearalenone in swine presents with distinct clinical signs reflecting excessive estrogenic stimulation. Affected prepubertal gilts exhibit pronounced vulvar swelling and hyperemia, mammary gland enlargement, and, in severe cases, vaginal or rectal prolapse. These signs result from the toxin binding to estrogen receptors, causing hypertrophy and hyperplasia of target reproductive tissues.

97. (B) Deoxynivalenol feed refusal toxin
In swine, the sudden onset of feed refusal and vomiting immediately after introducing a new grain batch is highly indicative of deoxynivalenol contamination. Also known as vomitoxin, this trichothecene mycotoxin is produced by Fusarium species and acts on the central nervous system to induce emesis and taste aversion, leading to significant production losses.

98. (D) Direct ionizing radiation exposure
The description of trichothecene mycotoxins as "radiomimetic" indicates that their cellular and systemic pathology closely mimics the lesions caused by direct ionizing radiation exposure. Both insults selectively target and destroy rapidly proliferating cell populations, including hematopoietic stem cells in bone marrow and crypt cells in the intestinal mucosa, resulting in pancytopenia and severe gastroenteritis.

99. (A) Severe oral mucosal ulceration and necrosis
Severe oral mucosal ulceration and necrosis is correct because T-2 toxin, a trichothecene mycotoxin produced by Fusarium species, is highly cytotoxic and primarily damages rapidly dividing epithelial cells. In poultry, acute toxicosis commonly causes erosions and necrotic ulcers in the oral cavity, reduced feed intake, and immunosuppression. The other lesions are characteristic of different toxic or metabolic conditions and are not typical features of acute T-2 toxin poisoning.

Downloaded from www.pdf.tube

154. (D) Hemoglobin is incapable of binding and transporting oxygen
Methemoglobinemia is a pathological condition characterized by the oxidation of the iron in hemoglobin from its functional ferrous state to the non-functional ferric state. In this altered state, the heme molecule is completely incapable of binding and transporting oxygen to peripheral tissues. This leads to severe cellular hypoxia, dark discoloration of blood, and clinical cyanosis.

155. (A) I, II and III
Cyanogenic glycoside toxicity occurs when livestock ingest plants like sorghum. Plant damage causes enzymic hydrolysis, releasing highly toxic hydrogen cyanide gas. Cyanide binds to the ferric iron of mitochondrial cytochrome c oxidase, blocking the electron transport chain and stopping cellular respiration. Affected blood remains fully oxygenated and is bright cherry-red, rather than dark chocolate-brown.

156. (C) Sodium nitrite and sodium thiosulfate therapy
Treating acute cyanogenic glycoside poisoning requires a dual therapeutic approach. First, sodium nitrite is administered intravenously to induce controlled methemoglobinemia, which competes with cytochrome c oxidase for cyanide ions, forming cyanmethemoglobin. Second, sodium thiosulfate is administered to provide a sulfur donor for the enzyme rhodanese, converting free cyanide into non-toxic, easily excreted thiocyanate.

157. (A) Bright cherry red
Cyanide poisoning blocks cellular oxygen utilization by inhibiting cytochrome c oxidase, while hemoglobin oxygen binding remains completely unaffected. Consequently, cells cannot extract oxygen from circulating blood, and oxygenated blood passes directly from arteries into the venous system. This results in highly oxygenated venous blood, giving it a characteristic and pathognomonic bright cherry-red color.

158. (D) Post-mortem blood is characteristically bright cherry red.
Nitrate/nitrite poisoning is defined by the oxidation of hemoglobin to methemoglobin. This pigment change turns the blood a characteristic dark chocolate-brown or muddy color. Methylene blue is the treatment of choice to reduce methemoglobin back to functional hemoglobin. Bright cherry-red post-mortem blood is characteristic of cyanide or carbon monoxide toxicity, not nitrate/nitrite poisoning.

159. (B) Dark, poorly coagulating chocolate-brown blood
The definitive pathological hallmark of acute nitrate toxicity in cattle is the appearance of the blood. During necropsy, the blood is characteristically dark, chocolate-brown, or muddy in color due to high concentrations of circulating methemoglobin. This altered blood also exhibits poor coagulation, and the tissues show generalized congestion and cyanosis due to systemic hypoxia.

160. (C) Renal system
Ingestion of of plants containing high concentrations of soluble oxalates, such as Halogeton, Sarcobatus, or Amaranthus, primarily targets the renal system. Once absorbed, soluble oxalates combine with systemic calcium ions to form insoluble calcium oxalate. These crystals precipitate directly within the renal tubules, causing severe mechanical damage to epithelial cells and leading to acute renal failure.

NODIA

161. (D) Hypocalcemia and renal calcium oxalate precipitation
The absorption of soluble oxalates from the digestive tract has dual, devastating pathophysiological effects. First, oxalates bind rapidly to circulating ionized calcium, causing sudden, severe hypocalcemia. Second, the resulting insoluble calcium oxalate complexes are filtered by the glomerulus and precipitate as crystals within the renal tubules, leading to mechanical damage and acute tubular necrosis.

162. (A) Acute renal tubular necrosis and gastroenteritis
Oak poisoning is a significant toxicosis of ruminants grazing Quercus species. The primary toxic principles are hydrolyzable tannins, including gallotannins and their metabolites like gallic acid. In the digestive tract, these compounds act as direct astringents, causing severe gastroenteritis. Upon systemic absorption, they cause severe, widespread damage to the kidneys, manifesting as acute renal tubular necrosis.

synthesis leading to vascular endothelial damage in the pulmonary vasculature. Gross examination of affected swine reveals heavy lungs with severe expansion of the interlobular septa by clear fluid and abundant frothy exudate within the trachea and airways.

110. (D) Rye and other cereal grasses
Ergotism is a historical and contemporary disease of animals caused by the ingestion of toxic ergot alkaloids. These toxic compounds are produced by the parasitic fungus Claviceps purpurea, which specifically infects the developing seed heads of rye, wheat, barley, and various other wild cereal grasses, replacing normal grains with dark sclerotia.

111. (B) Sustained peripheral vasoconstriction causing ischemic necrosis
The pathogenesis of gangrenous ergotism centers on the pharmacological activity of ergot alkaloids on blood vessels. These compounds act as alpha-adrenergic receptor agonists, stimulating prolonged, sustained vasoconstriction of arterioles and direct damage to vascular endothelium. This severe restriction of blood flow leads directly to peripheral ischemia, hypoxia, and subsequent dry gangrenous necrosis of distal extremities.

112. (C) Necrosis and sloughing of distal extremities
Degnala disease is a major toxicological condition affecting cattle and buffaloes, primary in South Asia. Pathologically and clinically, it closely mirrors classic gangrenous ergotism. The clinical presentation is defined by ischemic necrosis and subsequent dry sloughing of the tail, ears, and distal limbs, which occurs due to vasoconstrictive toxins on fungal-infected rice straw.

113. (D) Ergotamine - Ischemic dry gangrene
A clear cause-and-effect match exists between specific mycotoxins and their resulting pathology. Ingestion of the ergot alkaloid ergotamine causes sustained peripheral arterial vasoconstriction and endothelial cell damage. This severe lack of blood flow to the distal extremities leads to dry gangrene, manifesting as necrosis and sloughing of the distal limbs, tail, and ear tips.

114. (A) Ergotism
Fescue foot is a clinical syndrome of cattle grazing tall fescue infected with the endophytic fungus Neotyphodium coenophialum. This endophyte produces ergovaline, an ergot alkaloid that shares a direct toxicological mechanism with Claviceps purpurea. Both toxic agents stimulate peripheral alpha-adrenergic receptors, causing vasoconstriction that leads to dry gangrene, making the pathogenesis identical to ergotism.

115. (B) P-3, Q-4, R-1, S-2
Different mycotoxins cause distinct pathological syndromes. Ergotism leads to dry gangrene of extremities due to vasoconstriction. Zearalenone induces estrogenic effects like vulvar hypertrophy and prolapse. Equine fumonisin toxicosis results in liquefactive necrosis of cerebral white matter. Trichothecene toxicosis, being radiomimetic, damages rapidly dividing cells, causing mucosal necrosis of the gastrointestinal tract and lymphoid depletion.

116. (B) Cold ambient temperatures
The vasoconstrictive pathogenesis of gangrenous ergotism is significantly influenced by external climate factors. Low ambient temperatures induce physiological peripheral vasoconstriction to conserve core body heat. When this response is combined with the pharmacological vasoconstriction caused by ergot alkaloids, blood flow to distal tissues is completely halted, severely exacerbating tissue ischemia and dry gangrene.

117. (A) Cold, anesthetic, and sharply demarcated
Chronic gangrenous ergotism presents with distinct physical features in the extremities. Due to prolonged and complete lack of arterial blood supply, the affected distal parts, such as limbs, ears, and tail, become cold to the touch and lose all sensation. A clear, sharp line of demarcation develops between the necrotic and viable tissue before sloughing occurs.

118. (C) Synaptic acetylcholinesterase
Organophosphate and carbamate insecticides are potent neurotoxins used widely in agriculture and veterinary medicine. Their primary mechanism of acute toxicity involves targeting and binding to the enzyme synaptic acetylcholinesterase. By inhibiting this enzyme, they prevent the normal degradation of the neurotransmitter acetylcholine, leading to its accumulation in the synaptic cleft.

Click Here to Buy book on Flipkart

100. (C) Cytosolic estrogen receptors
Zearalenone is a structural analog of estrogen and exerts its toxic effects by binding specifically to cytosolic estrogen receptors in target tissues. This binding mimics the action of natural estradiol, triggering downstream gene transcription that leads to cellular hypertrophy and hyperplasia in the reproductive tract, manifesting as the classic hyperestrogenism syndrome in pigs.

101. (A) Fusarium verticillioides
Fumonisin toxicity is caused by mycotoxins produced primarily by Fusarium verticillioides, formerly known as Fusarium moniliforme, as well as Fusarium proliferatum. These fungi commonly infect corn and other cereal grains under specific environmental conditions. Ingestion of these contaminated grains leads to severe, species-specific diseases in livestock, including horses and swine.

102. (D) Altered sphingolipid pathway accumulating sphinganine
Fumonisin B1 is a structural analog of sphingoid bases and inhibits the enzyme ceramide synthase. This blockade prevents the synthesis of complex sphingolipids while causing a rapid accumulation of intracellular sphinganine and sphingosine, which are cytotoxic. This cellular disruption alters cell growth, differentiation, and membrane integrity, driving the pathogenesis of fumonisin-induced diseases.

103. (B) Liquefactive necrosis of equine cerebral white matter tracts
Equine leukoencephalomalacia is a highly fatal neurotoxic syndrome characterized by focal or diffuse liquefactive necrosis of the white matter tracts in the cerebral hemispheres of horses. This lesion results from ingesting fumonisin-contaminated corn, which disrupts sphingolipid metabolism and causes vascular damage in the brain, leading to severe edema, hypoxia, and tissue liquefaction.

104. (C) The lungs, inducing acute fatal pulmonary edema
The primary target organ of fumonisin toxicity in pigs is the respiratory system. Ingestion of fumonisin B1 causes porcine pulmonary edema, a disease characterized by the rapid accumulation of fluid within the pulmonary alveoli and interstitial spaces. This fatal condition results from altered sphingolipid metabolism leading to cardiovascular dysfunction and pulmonary vascular injury.

105. (C) A is true but R is false
Fumonisin exposure in pigs frequently leads to fatal pulmonary edema, making the assertion true. However, the reason provided is false, because fumonisin does not inhibit acetylcholinesterase or cause bronchoconstriction. Instead, its toxicity is driven by the inhibition of ceramide synthase, which alters sphingolipid pathways, leading to cardiovascular dysfunction and fluid accumulation in the lungs.

106. (D) Equine leukoencephalomalacia
Mycotoxic encephalomalacia occurs in horses consuming feed contaminated with fumonisin B1. This toxin is produced by Fusarium verticillioides, which typically infects corn. Clinical signs manifest as acute neurological deficits, including circling, head pressing, and blindness. The hallmark post-mortem finding consists of distinctive softening and liquefactive necrosis of the cerebral white matter.

NODIA

107. (A) Vascular endothelial integrity
Fumonisins exert toxicity by inhibiting the enzyme ceramide synthase, which disrupts the de novo biosynthesis of sphingolipids in cell membranes. The accumulation of sphingoid bases and depletion of complex sphingolipids lead to cell death and endothelial damage. Consequently, the primary pathophysiological outcome is the disruption of vascular endothelial integrity across various susceptible animal species.

108. (C) Severe immunosuppression - Sheep
Fumonisin toxicity displays clear species-dependent syndromes, such as equine leukoencephalomalacia and porcine pulmonary edema, along with hepatotoxicity in many domestic species. However, sheep do not exhibit severe immunosuppression as a characteristic clinical syndrome caused by this mycotoxin. Instead, ruminants generally possess high resistance to the toxic effects of fumonisin ingestion due to ruminal microbial degradation.

109. (A) Severe interlobular edema and frothy tracheal exudate
Porcine pulmonary edema is the classic manifestation of acute fumonisin toxicity in swine. The pathology is driven by altered sphingolipid

127. (B) Axonal sodium channels, causing neuroexcitation
Organochlorine insecticides, such as dichlorodiphenyltrichloroethane, are potent neurotoxic compounds. They exert their toxic effects primarily by interfering with the voltage-gated sodium channels along axonal membranes. By preventing the normal closure of these channels, organochlorines cause a prolonged sodium influx during action potentials. This leads to repetitive firing, membrane instability, and generalized central nervous system excitation.

128. (C) Tissue bioaccumulation and food chain biomagnification
Organochlorine compounds possess exceptional chemical stability and high lipophilicity. These physical properties make them highly resistant to environmental degradation and enzymatic metabolism. Once ingested, they readily distribute into adipose tissues, where they remain stored for long periods. This leads to progressive bioaccumulation in individual organisms and biomagnification to highly toxic concentrations up the food chain.

129. (D) Salivation, ear/paw flicking, tremors, and seizures
Pyrethroid toxicosis in cats commonly presents as a distinct, highly severe clinical syndrome. Cats are particularly susceptible because they lack efficient glucuronide conjugation pathways to detoxify these compounds. Affected felines manifest rapid-onset neurotoxicity characterized by excessive salivation, hyperesthesia, ear twitching, paw flicking, generalized muscle tremors, hyperthermia, and in severe cases, generalized tonic-clonic seizures.

130. (A) Absence of gross or microscopic lesions, requiring chemical assay
A significant diagnostic challenge in both organochlorine and pyrethroid toxicosis is the complete absence of pathognomonic gross or microscopic lesions. Animals that die from these neurotoxins show no structural tissue abnormalities on post-mortem or histological examination. Consequently, establishing a definitive diagnosis requires chemical analysis of adipose tissue, brain, liver, or stomach contents to detect toxic residues.

131. (B) Administration of muscle relaxants or anticonvulsants
Because pyrethroids cause prolonged opening of voltage-gated sodium channels, they produce repetitive nerve firing and severe, continuous muscular tremors. There is no specific chemical antidote for pyrethroid toxicosis. Therefore, clinical management focuses on supportive care, utilizing muscle relaxants like methocarbamol or anticonvulsants like diazepam to control tremors and stabilize the overexcited nervous system.

132. (B) Weight loss can trigger acute neurotoxicity from stored reserves
Due to their highly lipophilic nature, organochlorine compounds partition rapidly into body fat stores, where they remain biologically inactive. During periods of starvation, rapid weight loss, or negative energy balance, these fat reserves are mobilized. This process releases stored organochlorines back into the systemic circulation, allowing them to cross the blood-brain barrier and trigger acute neurotoxicity.

NODIA

133. (A) They cause profound inhibition of acetylcholinesterase.
Type II pyrethroids contain an alpha-cyano moiety, which makes them far more potent neurotoxins than Type I pyrethroids. They act by causing much longer delays in the inactivation of voltage-gated sodium channels and by inhibiting GABA-gated chloride channels. Unlike organophosphates and carbamates, pyrethroids do not possess any inhibitory action on the acetylcholinesterase enzyme.

134. (C) Cats
Permethrin is a common Type I synthetic pyrethroid widely utilized in canine ectoparasiticides. Cats are exceptionally sensitive to permethrin toxicity due to their deficient glucuronosyltransferase enzymes, which prevents efficient hepatic conjugation and clearance. Inappropriate application of canine-formulated permethrin products on cats regularly results in severe, life-threatening tremors, seizures, and marked hyperthermia.

135. (B) 2, 3, 1, 4
The toxicological pathway of anticoagulant rodenticide poisoning begins with oral ingestion of the active compound. Once absorbed, the toxin inhibits the crucial enzyme Vitamin K epoxide reductase, preventing the recycling

gossypol is not characterized by massive acute renal tubular necrosis, as its toxic effects are primarily directed at the myocardium and liver.

173. (A) Congestive heart failure and pulmonary edema
Monogastric animals, particularly young pigs, are highly susceptible to gossypol toxicosis. When fed un-detoxified cottonseed meal in high quantities, the cumulative toxin damages cardiac muscle cells. This leads to progressive myocardial degeneration and necrosis, manifesting clinically as bilateral congestive heart failure. Necropsy characteristically reveals a dilated, flabby heart, generalized venous congestion, and severe pulmonary edema.

174. (D) Ruminal proteins bind free gossypol, preventing absorption
Mature ruminants exhibit high resistance to gossypol compared to monogastrics and pre-ruminants. This tolerance is due to the activity of the rumen. Soluble proteins present in the ruminal fluid readily bind to free, active gossypol, converting it into a stable, insoluble complex. This complex cannot be absorbed, protecting the animal from systemic myocardial toxicity.

175. (A) Both A and R are true and R is the correct explanation of A.
Oleander leaves are extremely toxic, and ingestion of small amounts can cause sudden death in livestock. The plant contains highly active cardiac glycosides, primarily oleandrin, which inhibit the cellular Na^+/K^+ ATPase pump. This leads to severe intracellular calcium overload, causing fatal cardiac conduction block, severe ventricular arrhythmias, and sudden diastolic arrest.

176. (C) Dilated heart with pulmonary edema and hepatomegaly
The gross pathological findings of chronic gossypol toxicity are defined by congestive heart failure. Post-mortem examination characteristically reveals a globally dilated, hypertrophied, and flabby heart. This cardiac dysfunction leads to backward failure, causing severe pulmonary edema, diffuse congestion of the lungs, and passive hepatic congestion, presenting as hepatomegaly with a "nutmeg" liver appearance.

177. (D) A radiomimetic bone marrow suppressor and carcinogen
Pteridium aquilinum, or bracken fern, contains several toxic principles, most notably the norsesquiterpene glycoside ptaquiloside. This compound acts as a potent radiomimetic agent, directly damaging DNA in rapidly dividing cells. Consequently, it acts as a severe bone marrow suppressor, leading to aplastic anemia and pancytopenia, and acts as a primary carcinogen in various tissues.

178. (A) Widespread hemorrhages due to thrombocytopenia
Chronic bracken fern ingestion in cattle leads to the destruction of actively dividing hematopoietic cells within the bone marrow. This severe radiomimetic suppression results in pancytopenia. The profound depletion of circulating platelets (thrombocytopenia) destroys the blood's normal clotting ability, manifesting clinically and pathologically as widespread petechial and ecchymotic hemorrhages throughout all organs.

179. (B) Bladder bleeding from ptaquiloside-induced neoplasms
Enzootic bovine hematuria is a specific clinical syndrome of cattle caused by the chronic ingestion of low levels of bracken fern over several years. The carcinogen ptaquiloside induces the development of benign and malignant neoplasms within the urinary bladder wall, such as hemangiomas and carcinomas. These vascular tumors chronically bleed, resulting in persistent hematuria.

180. (B) Thiaminase-induced thiamine deficiency and polioencephalomalacia
In equines, bracken fern toxicity presents as a neurological syndrome rather than bone marrow suppression. Bracken fern contains highly active thiaminase enzymes that destroy dietary thiamine within the gastrointestinal tract. This leads to a profound systemic thiamine (Vitamin B1) deficiency, which disrupts brain energy metabolism and causes classic polioencephalomalacia, presenting as ataxia and tremors.

181. (A) Intravenous thiamine supplementation
Horses presenting with progressive ataxia, incoordination, and muscle tremors after consuming bracken fern are suffering from thiaminase-induced thiamine deficiency. Because the neurological lesions of polioencephalomalacia

Downloaded from www.pdf.tube

of unpigmented skin, known as hepatogenous photosensitization.

145. (D) I and II
Pyrrolizidine alkaloid toxicosis is a major cause of chronic liver disease in livestock. These alkaloids undergo metabolic activation in the liver to highly reactive pyrroles. These pyrroles damage DNA, leading to inhibition of mitosis. Affected hepatocytes can still synthesize DNA but cannot divide, resulting in hepatocyte megalocytosis. This progressive damage culminates in hepatic fibrosis and cirrhosis.

146. (A) Dermatitis from phylloerythrin accumulation reacting with light
Hepatogenous photosensitization occurs when a primary hepatic lesion, such as cholestasis or biliary obstruction, impairs the excretion of phylloerythrin. This green plant pigment byproduct accumulates in the systemic vasculature of the skin. When exposed to ultraviolet light, phylloerythrin is photo-activated, releasing energy that causes localized severe dermatitis and necrosis of non-pigmented epidermis.

147. (B) Lantana camara
An animal displaying marked icterus, photophobia, and dry sloughing of unpigmented epidermal areas has hepatogenous photosensitization. The primary suspect for this presentation is Lantana camara, which contains lantadenes that cause intrahepatic cholestasis. The resulting failure to excrete phylloerythrin leads to its accumulation in skin capillaries, where solar exposure triggers severe cutaneous phototoxic damage.

148. (B) Crotalaria - Megalocytosis and veno-occlusion
Hepatotoxic plants produce highly specific lesions that allow diagnostic differentiation. Plants of the genus Crotalaria contain pyrrolizidine alkaloids. These compounds are metabolized in hepatocytes to reactive pyrroles that inhibit mitosis, resulting in the classic histologic hallmarks of hepatocyte megalocytosis. In some species, they also cause pulmonary congestion and hepatic veno-occlusive disease.

149. (A) Synthesize DNA but fail to divide, forming megalocytes
Pyrrolizidine alkaloids undergo biotransformation in the liver to active pyrrole metabolites. These metabolites act as potent alkylating agents that bind to cellular DNA, causing irreversible cross-linking. This molecular damage blocks mitosis while leaving DNA replication intact. Consequently, affected hepatocytes continue to synthesize DNA and grow in size but cannot divide, forming classic hypertrophied megalocytes.

150. (C) Acute massive renal hemorrhage
Chronic pyrrolizidine alkaloid toxicosis presents with progressive lesions centered in the liver. Pathological features include extensive hepatic fibrosis, biliary hyperplasia, and megalocytosis, which leads to portal hypertension, ascites, and hepatic encephalopathy from elevated blood ammonia. However, acute massive renal hemorrhage is not a characteristic pathological feature of this chronic hepatotoxic plant poisoning.

151. (D) Senecio jacobaea
Senecio jacobaea, commonly known as tansy ragwort, is a highly significant toxic plant of grazing livestock. This species contains abundant pyrrolizidine alkaloids that undergo hepatic biotransformation to toxic pyrroles. Ingestion of this plant is a classic cause of chronic hepatotoxicosis, characterized histologically by hepatocyte megalocytosis, diffuse bridging fibrosis, and bile duct hyperplasia.

152. (C) Cyanide
Nitrate poisoning is closely related to the accumulation of toxic compounds in the rumen. While nitrate is classically reduced to nitrite, some pathway cross-talk or historical classifications associated toxic plant syndromes with both nitrite and cyanide production. Cyanide release occurs in ruminants consuming cyanogenic plants, which blocks cellular respiration by binding to cytochrome c oxidase.

NODIA

153. (B) Oxidizing hemoglobin to form methemoglobin
Absorbed nitrite ions react directly with the iron in circulating hemoglobin. Nitrite oxidizes the normal ferrous iron ($Fe^{\{2+\}}$) to the ferric state ($Fe^{\{3+\}}$), forming methemoglobin. Ferric iron is incapable of binding and transporting oxygen to tissues. This alteration results in profound systemic hypoxia and cyanosis, which is the primary mechanism of nitrite-induced lethal toxicity.

119. (D) Synaptic accumulation and overstimulation of acetylcholine
The direct pharmacological effect of acetylcholinesterase inhibition by organophosphates is the persistent accumulation of acetylcholine. Because the physiological inactivation pathway of this neurotransmitter is blocked, acetylcholine accumulates in high concentrations within both muscarinic and nicotinic synaptic junctions. This causes continuous, repetitive overstimulation of the postsynaptic receptors, leading to cholinergic crisis.

120. (C) SLUDGE syndrome signs including bradycardia
Muscarinic signs in organophosphate toxicosis are produced by the hyperstimulation of postganglionic parasympathetic nerve terminals. These manifestations are commonly grouped under the SLUDGE acronym, which stands for salivation, lacrimation, urination, defecation, gastrointestinal hypermotility, and emesis. Bradycardia, miosis, and bronchoconstriction are also prominent muscarinic responses that occur during this clinical presentation.

121. (B) Carbamates require massive Phase I activation to become toxic.
Organophosphates and carbamates differ fundamentally in their interaction with acetylcholinesterase. Organophosphates form a stable, covalent bond that can undergo irreversible "aging," whereas carbamates produce a reversible carbamylation of the enzyme that spontaneously dissociates. However, carbamates do not require massive Phase I metabolic activation to become toxic, as they are inherently active as direct inhibitors.

122. (D) Administration of Atropine to block muscarinic receptors
A patient presenting with acute parasympathetic overstimulation, including miosis, severe salivation, lacrimation, defecation, and muscle tremors, requires immediate pharmacological intervention. Atropine sulfate is the specific competitive antagonist for muscarinic acetylcholine receptors. Administering atropine blocks the excessive accumulation of acetylcholine at these receptor sites, effectively reversing life-threatening bradycardia, bronchoconstriction, and excessive respiratory secretions.

123. (A) Respiratory failure from paralysis and secretions
In fatal cases of organophosphate or carbamate poisoning, death is primarily due to respiratory failure. The massive accumulation of acetylcholine causes continuous stimulation of muscarinic and nicotinic receptors. This overstimulation leads to a depolarization block of the diaphragm and intercostal muscles, which is severely compounded by bronchoconstriction and excessive bronchial secretions blocking the airways.

124. (C) Contaminated municipal water supplies
Veterinary exposure to organophosphate and carbamate insecticides typically occurs through contact with agricultural crop sprays, veterinary ectoparasitic treatments like flea and tick dips, or accidental ingestion of concentrated formulations. Contaminated municipal water supplies are highly regulated and monitored, making them an extremely rare and highly unlikely source of toxicity in domestic animal populations.

125. (A) 1. OP binds acetylcholinesterase → 2. Acetylcholine accumulates → 3. Receptor overstimulation → 4. Depolarization block and paralysis
The pathogenesis of acute organophosphate poisoning follows a precise molecular sequence. First, the organophosphate compound binds covalently to the active site of acetylcholinesterase. This inhibition prevents acetylcholine hydrolysis, causing its rapid accumulation in the synaptic cleft. The accumulated neurotransmitter overstimulates postsynaptic receptors, ultimately leading to a persistent depolarization block and flaccid paralysis.

126. (D) Reduced cholinesterase in blood or brain tissue
Confirming organophosphate poisoning at necropsy or clinically is challenging because these compounds do not produce specific, characteristic gross pathological lesions. Therefore, diagnostic confirmation depends on demonstrating a significant reduction in cholinesterase enzyme activity. Assaying whole blood, plasma, or post-mortem brain tissue for inhibited cholinesterase provides direct, reliable evidence of exposure and subsequent toxicosis.

Click Here to Buy book on Amazon

163. (B) Birefringent rosette-shaped crystals in tubular lumens
The histopathological diagnosis of oxalate poisoning relies on identifying calcium oxalate crystals within the kidney. On microscopic examination, these crystals appear in the lumens of the proximal and distal tubules as distinctive rosette-shaped, colorless, and highly birefringent structures under polarized light. This presentation is accompanied by severe epithelial degeneration, necrosis, and intratubular casts.

164. (B) Oak poisoning
A cow displaying extensive perirenal edema, pale swollen kidneys with petechiae, and severe mucoid hemorrhagic enteritis after grazing in areas abundant with Quercus species is affected by oak poisoning. The gallotannins present in oak leaves and acorns cause direct chemical damage to the vascular endothelium and epithelial cells of both the gastrointestinal tract and renal tubules.

165. (A) I, II, III and IV
Toxic nephrosis is caused by several plant species. Pigweed induces marked perirenal edema and tubular necrosis. Ruminants can tolerate moderate oxalate intake due to degradation by Oxalobacter formigenes. Tannins in oak leaves cross-link cell proteins, causing necrosis, while oxalate crystals cause physical tubular damage. Oak toxicity does not spare the gastrointestinal tract, causing severe mucoid enteritis.

166. (C) Acute hypocalcemic tremors and tetany
In the acute phase of soluble oxalate poisoning, clinical signs manifest very rapidly. This rapid clinical onset is driven primarily by the sudden, massive binding of oxalates to systemic calcium. This precipitous drop in serum ionized calcium induces profound hypocalcemia, leading to neuromuscular excitability, muscle tremors, tetany, weakness, and collapse before renal lesions have time to develop.

167. (D) It causes acute massive hepatic necrosis as its primary lesion.
Oak poisoning in livestock is defined by its primary effects on the kidneys and gastrointestinal tract. Young buds and green acorns contain high levels of gallotannins, which cause severe renal tubular necrosis and ulcerative gastrointestinal lesions. However, oak poisoning is not characterized by acute massive hepatic necrosis, as its primary target organs are the renal tubules and intestinal mucosa.

168. (C) Ruminal Oxalobacter formigenes degrades oxalates
Ruminants possess a marked capacity to adapt to diets containing moderate levels of soluble oxalates. This resistance is due to the presence of specific anaerobic bacteria in the rumen, primarily Oxalobacter formigenes. These specialized microbes utilize oxalate as a primary energy source, degrading it into non-toxic formate and carbon dioxide, thereby preventing systemic absorption and subsequent toxicity.

169. (B) Cardiac glycosides
Nerium oleander is a highly toxic ornamental shrub that is extremely dangerous to all animal species. All parts of the plant contain high levels of cardiotoxic compounds, primarily oleandrin and neriine. These compounds are classified as cardiac glycosides, which act directly on the myocardium to disrupt electrical conduction and cause sudden death.

170. (D) Increased intracellular calcium causing severe arrhythmias
Cardiac glycosides exert toxicity by binding to and inhibiting the membrane-bound Na^+/K^+ ATPase pump. This inhibition increases intracellular sodium, which in turn reduces the activity of the Na^+/CA^{2+} exchanger. Consequently, intracellular calcium levels rise significantly. This calcium overload increases myocardial contractility but also triggers severe, life-threatening cardiac arrhythmias and diastolic arrest.

171. (A) Cotton
Gossypol is a natural polyphenolic pigment compound produced by the pigment glands of plants belonging to the genus Gossypium. This toxin is primarily concentrated in the seeds of the cotton plant. Cottonseed meal is widely used as a protein supplement in livestock feeds, which frequently leads to accidental toxicosis when fed to sensitive animal species.

NODIA

172. (C) Massive acute renal tubular necrosis
Different cardiotoxins cause highly specific syndromes. Oleandrin induces severe cardiac arrhythmias. Taxine blocks conduction, causing sudden cardiac arrest. Gossypol is a cumulative cardiotoxin that causes myocardial degeneration, necrosis, and congestive heart failure. However,

of inactive Vitamin K. Consequently, active Vitamin K-dependent clotting factors are progressively depleted, ultimately resulting in severe, widespread internal and external hemorrhage.

136. (C) II, VII, IX, X
Anticoagulant rodenticides act by inhibiting Vitamin K epoxide reductase, which prevents the essential carboxylation of specific coagulation factors. These critical, Vitamin K-dependent clotting factors are Factor II (prothrombin), Factor VII, Factor IX, and Factor X. Depletion of these functional proteins disrupts both intrinsic and extrinsic coagulation cascades, resulting in severe clotting failure.

137. (B) Hypercalcemia causing widespread tissue mineralization
Cholecalciferol rodenticides cause severe toxicity by promoting the massive gastrointestinal absorption and renal reabsorption of calcium. This results in profound, life-threatening hypercalcemia and concurrent hyperphosphatemia. High circulating levels of these minerals exceed the solubility product, causing metastatic calcification and widespread mineralization of soft tissues, particularly the kidneys, gastrointestinal tract, and cardiac muscle.

138. (D) Phosphine gas
Zinc phosphide is a highly toxic rodenticide that, upon ingestion, reacts rapidly with water and hydrochloric acid in the gastric environment. This chemical reaction liberates phosphine gas, which is highly volatile and toxic. Phosphine gas is absorbed systemically, where it acts as a cellular poison by inhibiting cytochrome c oxidase and inducing severe oxidative damage.

139. (A) Anticoagulant rodenticide
A patient presenting with acute dyspnea, pale mucous membranes, and cavitary effusions days after potential toxin exposure points strongly toward anticoagulant rodenticide toxicosis. Clotting factor depletion requires several days to manifest clinically. The hemorrhage frequently presents as hemothorax, mediastinal hematomas, or hemoabdomen, which directly cause respiratory distress and severe blood-loss anemia.

140. (C) Active phosphine gas poses a severe inhalation risk
Detecting a "garlic" or "rotten fish" odor during the necropsy of an animal suspected of zinc phosphide poisoning indicates the active release of phosphine gas. This gas is highly toxic to humans and animals. It poses an immediate, severe inhalation hazard to the pathologist and veterinary staff, requiring prompt ventilation and safety precautions.

141. (A) Massive uncoagulated blood in the thorax
Cholecalciferol toxicity causes metastatic calcification of tissues due to severe hypercalcemia. Hallmark post-mortem findings include chalky mineral plaques on the epicardium, a gritty texture when sectioning the kidneys, and mineralization of the gastric mucosa and blood vessels. Massive uncoagulated blood in the thorax is characteristic of anticoagulant rodenticides, not cholecalciferol toxicosis.

142. (D) Prolonged therapy with oral Vitamin K1
Second-generation anticoagulant rodenticides, such as brodifacoum, possess exceptionally long biological half-lives. Once ingested, they persist in hepatic tissues and inhibit clotting factor synthesis for several weeks. Consequently, treating exposed veterinary patients requires prolonged, consistent therapy with oral Vitamin K1, often spanning three to four weeks, to maintain functional coagulation factors.

143. (B) A hepatotoxin causing intrahepatic cholestasis
Lantana camara is a widespread, highly toxic ornamental plant. Ruminants are especially susceptible to its active triterpene acids, primarily lantadene A and B. These toxic compounds specifically target the liver, where they cause direct damage to canalicular membranes. This damage results in severe, progressive intrahepatic cholestasis and subsequent systemic accumulation of bile constituents.

144. (C) Hepatogenous photosensitization
Intrahepatic cholestasis induced by Lantana camara prevents the normal biliary excretion of phylloerythrin, a photodynamic product of chlorophyll breakdown. Consequently, phylloerythrin accumulates in the systemic circulation and skin capillaries. Upon exposure to sunlight, this compound absorbs UV light and generates free radicals, causing severe necrosis

Downloaded from www.pdf.tube

are caused by altered cellular energy metabolism, the most effective and logical treatment is immediate, high-dose intravenous thiamine supplementation, which rapidly reverses clinical signs if administered early.

182. (C) Immunosuppression and infection from leukopenia
The radiomimetic mechanism of ptaquiloside in cattle targets all rapidly dividing cell populations. Alongside severe thrombocytopenia and resulting hemorrhage, the bone marrow suppression destroys the production of white blood cells. This severe leukopenia, particularly neutropenia, causes marked systemic immunosuppression. Consequently, affected animals are highly vulnerable to secondary, opportunistic bacterial infections and septicoses.

183. (D) Spastic paresis of the hindlimbs
Bracken fern toxicity manifests as distinct syndromes. In cattle, chronic exposure causes bone marrow suppression, leading to hemorrhagic diathesis, enzootic hematuria, and urinary bladder tumors. Spastic paresis of the hindlimbs is a separate hereditary or developmental neuromuscular disorder of cattle that is completely unrelated to the radiomimetic or toxic effects of bracken fern.

184. (C) Widespread hemorrhages and luminal intestinal blood
The gross post-mortem lesions of acute bracken fern toxicity in cattle reflect severe pancytopenia and vascular damage. Necropsy reveals widespread petechial, ecchymotic, and suffusive hemorrhages across all internal organs, serosal surfaces, and mucous membranes. Large quantities of free, uncoagulated blood are commonly found within the lumen of the small and large intestines.

185. (B) P-2, Q-3, R-1, S-4
Different toxic plants cause classic, well-documented pathological outcomes. Bracken fern consumption leads to enzootic bovine hematuria via bladder neoplasia. Oleander ingestion causes sudden death through cardiac glycosides blocking conduction. Sorghum grazing releases hydrogen cyanide, causing peracute cellular hypoxia. Lantana camara induces intrahepatic cholestasis, preventing phylloerythrin excretion and culminating in hepatogenous photosensitization.

186. (D) Allow rumen microbes to synthesize protein
Non-protein nitrogen (NPN) sources, such as urea, are added to ruminant feeds as a cost-effective nitrogen supplement. Ruminants cannot utilize urea directly. Instead, rumen microflora hydrolyze urea to ammonia, which they then use alongside fermentable carbohydrates to synthesize high-quality microbial protein. This microbial protein is subsequently digested and absorbed in the lower tract.

187. (A) Ammonia
When ruminants ingest large quantities of urea, the rate of hydrolysis in the rumen is extremely rapid. The urease enzyme produced by rumen microflora quickly breaks down urea, releasing large amounts of ammonia. If the release of ammonia exceeds the microbes' capacity to utilize it for protein synthesis, it accumulates within the rumen.

NODIA

188. (C) Systemic hyperammonemia exceeding hepatic urea cycle capacity
The fundamental pathogenesis of NPN toxicity is systemic hyperammonemia. When ruminal ammonia levels rise rapidly, the gas is absorbed into the portal circulation. Once in the liver, ammonia is converted back to urea. However, when portal absorption exceeds the hepatic urea cycle's maximum capacity, free ammonia escapes into systemic circulation, causing profound neurotoxicity.

189. (A) Ruminal cold water and vinegar infusion to lower pH
Treating acute urea toxicosis in cattle requires shifting the chemical equilibrium of ammonia in the rumen. Administering an infusion of cold water and vinegar directly into the rumen lowers the pH. This acidic environment converts highly volatile, lipophilic ammonia (NH_3) into the charged, non-absorbable ammonium ion (NH_4^+), while the cold temperature slows bacterial urease activity.

190. (D) Rise significantly, often exceeding 8.0
Free ammonia is highly alkaline in solution. During acute urea toxicosis, the massive and rapid generation of ammonia within the rumen exceeds the buffering capacity of the ruminal fluid. This chemical shift causes the ruminal pH to rise significantly from its normal neutral range, frequently exceeding 8.0, which further facilitates the absorption of non-ionized ammonia.

(B) Multifocal miliary hepatic necrosis
(C) Severe chronic caseous pneumonia
(D) Acute purulent leptomeningitis

2001

8. What is the most appropriate diagnostic method to detect Syphacia obvelata infection in a laboratory mouse colony?
(A) Fecal flotation using saturated sodium chloride solution
(B) Deep skin scrapings of the dorsal neck region
(C) Cellophane tape test applied to the perianal region
(D) Direct smear of duodenal contents

2019

9. Chronic progressive nephropathy in aging Sprague-Dawley rats is most heavily exacerbated by which dietary factor?
(A) High carbohydrate intake
(B) High fat intake
(C) Low calcium intake
(D) High protein intake

2014

10. Given below are two statements: one is asserted as Assertion (A) and the other is labeled as Reason (R).
Assertion (A): Infection with Rabbit hemorrhagic disease virus (RHDV) typically spares rabbits younger than 4 to 8 weeks of age.
Reason (R): Young rabbits possess passive maternal antibodies that neutralize the virus completely before it can attach to hepatocytes.
(A) Both (A) and (R) are true and (R) is the correct explanation of (A).
(B) Both (A) and (R) are true but (R) is not the correct explanation of (A).
(C) (A) is true but (R) is false.
(D) (A) is false but (R) is true.

2013

11. Which bacterial pathogen is the primary etiological agent of "Snuffles" in rabbits, often leading to mucopurulent rhinitis and pneumonia?
(A) Bordetella bronchiseptica
(B) Pasteurella multocida
(C) Streptococcus pneumoniae
(D) Staphylococcus aureus

2010

12. Scurvy in guinea pigs results from an absolute dietary deficiency of Vitamin C. This deficiency directly impairs which biochemical process?
(A) Gamma-carboxylation of clotting factors in the liver
(B) Proline and lysine hydroxylation in collagen synthesis
(C) Conversion of cholecalciferol to its active dihydroxy form
(D) Active absorption of iron from the gastrointestinal tract

2016

13. Which of the following constitutes the most relevant gross pathological finding in a rhesus macaque naturally infected with Mycobacterium tuberculosis?
(A) Caseous granulomas in the lungs and bronchial lymph nodes
(B) Diffuse mucosal thickening and corrugations of the distal ileum
(C) Multiple large vegetative lesions on the mitral heart valve
(D) Severe acute bilateral adrenal gland hemorrhage and necrosis

2012

14. Which finding is the most reliable indicator of Canine Distemper Virus infection in a wild tiger during post-mortem histopathology?
(A) Eosinophilic intracytoplasmic inclusions within neurons of the hippocampus
(B) Large basophilic intranuclear inclusions inside hepatic parenchymal cells
(C) Intracytoplasmic and intranuclear inclusions in respiratory epithelium
(D) Eosinophilic intracytoplasmic Negri bodies in cerebellar Purkinje cells

2025

15. Identify the INCORRECT pair regarding parasitic diseases in wild carnivores and their primary pathological effect.
(A) Dirofilaria immitis - Right-sided congestive heart failure
(B) Babesia canis - Intravascular hemolysis
(C) Sarcoptes scabiei - Hyperkeratotic dermatitis
(D) Trypanosoma evansi - Severe purulent encephalitis

2008

16. In wild ruminants, an outbreak characterized by erosive stomatitis, severe diarrhea, and high mortality, especially in younger animals, strongly suggests infection with which of the following agents?
(A) Foot-and-mouth disease virus
(B) Mycobacterium avium subspecies paratuberculosis
(C) Peste des Petits Ruminants virus

Click Here to Buy book on Flipkart

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

Heinz body anemia. However, this susceptibility is not due to high glucuronyl transferase activity. Instead, cats are deficient in this crucial glucuronidation enzyme, preventing normal metabolism. Consequently, acetaminophen is shunted to Phase I oxidation, generating highly reactive metabolites that oxidize erythrocyte proteins.

201. (B) Severe generalized exostoses
NSAID toxicosis in dogs presents with distinct pathological lesions centered in the gastrointestinal tract and kidneys. Common findings include gastric erosion and perforating ulcers, renal crest or papillary necrosis, and hypoalbuminemia secondary to protein-losing enteropathy. Severe generalized exostoses (bony growths) are not associated with NSAID toxicity, representing chronic vitamin A toxicosis or metabolic bone disease.

202. (D) Diclofenac
The appearance of chalky, white uric acid deposits on the serosal surfaces of internal organs, known as visceral gout, is the classic pathological hallmark of renal failure in birds. In vulture populations, this presentation is highly indicative of exposure to diclofenac, an NSAID that causes severe renal ischemia and acute tubular necrosis.

203. (A) Kidneys and inner ear (nephrotoxicity and ototoxicity)
Aminoglycoside antibiotics, such as gentamicin, neomycin, and amikacin, are highly effective against Gram-negative bacteria but have a narrow safety margin. They selectively accumulate within the cells of two primary organ systems, leading to severe toxic manifestations. These classic targeted toxicities are acute renal tubular necrosis (nephrotoxicity) and destruction of hair cells in the inner ear (ototoxicity).

204. (C) Massive necrosis of skeletal and cardiac muscle
Ionophore antibiotics, such as monensin, act by facilitating the transport of sodium ions across biological membranes. This cellular influx disrupts electrochemical gradients and promotes an influx of calcium ions into the cytosol. This severe calcium overload damages mitochondrial membranes and activates proteolytic enzymes, leading directly to massive necrosis of skeletal and cardiac muscle fibers.

205. (A) P-3, Q-2, R-1, S-4
Different veterinary drugs and chemicals produce distinctive pathological hallmarks. Aminoglycosides cause acute tubular necrosis without crystal formation. Ionophores lead to severe myocardial and skeletal muscle necrosis via calcium overload. Ethylene glycol ingestion results in calcium oxalate crystal nephropathy. Acetaminophen exposure in cats causes Heinz body hemolytic anemia and methemoglobinemia due to glucuronidation deficiency.

206. (D) Calcium oxalate crystals
Ethylene glycol poisoning is a peracute toxicosis of dogs and cats. Once ingested, ethylene glycol is metabolized by alcohol dehydrogenase to glycolic acid and subsequently oxalic acid. Oxalic acid combines with circulating calcium, forming insoluble calcium oxalate. These crystals precipitate as birefringent prisms in the renal tubules, causing severe mechanical epithelial damage and acute renal failure.

207. (B) Inhibit alcohol dehydrogenase, preventing toxic metabolites
The parent compound, ethylene glycol, is relatively non-toxic. The severe clinical and pathological effects are caused by its highly acidic metabolites, including glycolic and oxalic acids. Administering fomepizole or ethanol competitively inhibits the enzyme alcohol dehydrogenase. This blockade prevents the biotransformation of ethylene glycol, allowing the parent compound to be excreted safely and unchanged.

208. (C) Creatine kinase (CK) and cardiac troponins
Severe muscle damage is the defining pathological hallmark of ionophore toxicity in equines. The massive cellular calcium overload triggers widespread necrosis of both skeletal and cardiac myocytes. Consequently, biochemical analysis of serum from affected horses reveals dramatic, rapid elevations of creatine kinase, aspartate aminotransferase, and highly specific markers of myocardial injury like cardiac troponins.

209. (D) Cattle
Susceptibility to ionophore toxicity varies significantly among species. Horses are notoriously the most sensitive species, with a highly lethal dose of monensin. Poultry and dogs also exhibit high susceptibility. In contrast, mature cattle are relatively resistant and

Downloaded from www.pdf.tube

transport
(B) Replication in salivary glands → CNS replication → Centripetal axonal transport → Viral replication in myocytes
(C) CNS replication → Viral replication in myocytes → Centrifugal axonal transport → Replication in salivary glands
(D) Viral replication in myocytes → Centripetal axonal transport → CNS replication → Centrifugal axonal transport to salivary glands

2013
26. Which of the following statements is INCORRECT regarding the clinical presentation and pathology of Salmonellosis in laboratory mice?
(A) Severe acute diarrhea is consistently observed in all infected strains.
(B) Infection often remains subclinical in resistant mouse strains.
(C) Microscopic lesions typically include thrombosis of hepatic venules.
(D) The spleen frequently exhibits marked enlargement and histiocytic infiltration.

2004
27. The development of amyloidosis in aging laboratory mice is most frequently classified under which of the following amyloid types?
(A) AL amyloid
(B) AA amyloid
(C) Aβ amyloid
(D) ATTR amyloid

2019
28. In free-ranging wild animals, legal forensic sampling for suspected organophosphate poisoning must prioritize which specimen for the detection of cholinesterase inhibition?
(A) Hair follicles, fur, and claws
(B) Whole blood and brain tissue
(C) Feces and urine
(D) Bone marrow and spleen

2003
29. Identify the correct statement regarding Kyasanur Forest Disease.
(A) It is a tick-borne viral hemorrhagic fever affecting both monkeys and humans.
(B) It is primarily a respiratory illness transmitted via aerosolized rodent feces.
(C) It causes high mortality in wild birds, serving as a sentinel for human outbreaks.
(D) It is caused by a Bunyavirus endemic to the Western Himalayas.

NODIA

2012
30. Consider the following statements regarding diseases of guinea pigs:
I. Pregnancy toxemia in guinea pigs is characterized by severe hepatic lipidosis.
II. Vitamin C deficiency causes defective osteoid formation resulting in enlarged costochondral junctions.
III. Cytomegalovirus strictly causes fatal systemic disease in all adult guinea pigs.
Which of the above statement(s) is/are correct?
(A) I and II
(B) I
(C) II and III
(D) I, II and III

2022
31. What is the primary disadvantage of using gross pathology alone to diagnose Peste des Petits Ruminants in wild herbivores?
(A) The virus causes no discernible gross pathological lesions in infected wild ruminants.
(B) Gross pathology only reveals localized lesions in the central nervous system, completely missing systemic signs.
(C) The rapid onset of autolysis in lung tissue completely obscures any pathognomonic lesions.
(D) Gross lesions are identical to Rinderpest, requiring molecular differentiation.

2002
32. In laboratory rats, Sendai virus infection most commonly results in which type of pathological change?
(A) Severe acute hemorrhagic cystitis with hematuria
(B) Multifocal demyelinating encephalomyelitis in the brainstem
(C) Interstitial pneumonia and bronchiolar necrosis
(D) Chronic granulomatous hepatitis with focal necrosis

2009
33. Based on the pathogenesis of Diclofenac toxicity in vultures, which physiological process is directly inhibited, leading to fatal urate diathesis?
(A) Uricase enzyme activity in the liver
(B) Renal tubular excretion of uric acid
(C) Glomerular filtration due to renal portal vein thrombosis

CHAPTER 14

Pathology of Diseases of Laboratory and Wild Animals

2022

1. Which of the following best defines a gnotobiotic animal in the context of laboratory animal pathology?
(A) An animal derived by cesarean section and maintained in a sterile environment with no demonstrable microorganisms.
(B) An animal that harbors a known, defined microflora and fauna, and is free of all other demonstrable microorganisms.
(C) An animal specifically bred to be free of particular pathogens that commonly affect that species.
(D) An animal raised in an open environment but regularly tested to ensure the absence of zoonotic pathogens.

2021

2. When conducting a medicolegal post-mortem examination on a free-ranging tiger suspected of being poisoned, which tissue sample is LEAST relevant for primary toxicological analysis?
(A) Stomach contents
(B) Liver parenchyma
(C) Adipose tissue
(D) Synovial fluid

2014

3. What is the most common physiological cause of naturally occurring elevated alkaline phosphatase in growing specific pathogen-free (SPF) Wistar rats?
(A) Rapid osteoblastic activity
(B) Hepatic lipidosis
(C) Renal tubular degeneration
(D) Biliary stasis

2015

4. Identify the INCORRECT statement regarding occupational health hazards in laboratory animal facilities.
(A) Macacine herpesvirus 1 is a significant occupational hazard that can cause fatal encephalomyelitis in humans.
(B) Lymphocytic choriomeningitis virus is typically transmitted to humans via aerosolized rodent urine.
(C) Q fever in personnel is most commonly associated with exposure to infected pregnant lagomorphs.
(D) Laboratory animal allergy is one of the most common occupational risks for personnel handling rodents.

2015

5. In laboratory mice, Mousepox is characterized by primary viral replication in which of the following initial anatomical sites?
(A) Regional lymph nodes following skin inoculation
(B) Respiratory epithelium and alveolar macrophages
(C) Mucosal lining of the gastrointestinal tract
(D) Ependymal cells of the central nervous system

2011

6. Which of the following is an advantage of utilizing specific pathogen-free animals rather than germ-free animals in nutritional pathology research?
(A) They possess a completely sterile gastrointestinal tract, preventing microbial fermentation of the diet.
(B) They have an underdeveloped immune system, which accentuates nutritional deficiencies.
(C) They require less rigorous biosecurity measures than conventionally housed animals.
(D) They exhibit normal gastrointestinal tract morphology and physiological absorption rates.

2005

7. Tyzzer's disease in laboratory rodents, caused by Clostridium piliforme, produces distinct gross lesions. Which of the following correctly pairs the pathogen with its pathognomonic lesion?
(A) Acute diffuse hemorrhagic cystitis

Click Here to Buy book on Amazon

frequently receive monensin in their diets as a growth promoter and coccidiostat, tolerating far higher doses.

210. (A) P-3, Q-1, R-4, S-2
Antidotal therapy is highly specific for different toxic agents. Organophosphates are treated with atropine to block muscarinic receptors and pralidoxime to reactivate acetylcholinesterase. Ethylene glycol ingestion requires fomepizole or ethanol to inhibit alcohol dehydrogenase. Acetaminophen toxicity is managed with N-acetylcysteine to restore glutathione levels. Anticoagulant rodenticide poisoning requires prolonged Vitamin K1 therapy.

****

NODIA

(D) Pasteurella multocida

2019

17. The Elephant Endotheliotropic Herpesvirus (EEHV) primarily targets which specific cell type, leading to fatal hemorrhagic disease in young Asian elephants?
(A) Epidermal keratinocytes in the skin
(B) Capillary endothelial cells
(C) Hepatic parenchymal cells
(D) Pulmonary alveolar macrophages

2008

18. An outbreak of high mortality in wild water birds involves gross lesions of petechial hemorrhages on the epicardium, congested lungs, and necrosis of the pancreas. Which viral etiology is the most logical cause?
(A) Avian Poxvirus infection
(B) Infectious Bursal Disease virus infection
(C) Marek's Disease virus strain
(D) Highly Pathogenic Avian Influenza

2014

19. Visceral gout in captive raptors is typically an outcome of impaired clearance of which metabolic byproduct?
(A) Uric acid
(B) Urea
(C) Ammonia
(D) Creatinine

2025

20. Match the captive reptile diseases in List I with their characteristic pathological features in List II.

List I: (Diseases)
P. Infectious stomatitis
Q. Inclusion body disease
R. Nutritional secondary hyperparathyroidism
S. Gout

List II: (Pathological Features)
1. Fibrous osteodystrophy
2. Eosinophilic intracytoplasmic inclusions in neurons
3. Petechiae and caseous exudate in the oral cavity
4. Tophi deposition in joints and viscera

(A) P-3, Q-1, R-2, S-4
(B) P-3, Q-2, R-1, S-4
(C) P-4, Q-2, R-1, S-3
(D) P-1, Q-3, R-4, S-2

2007

21. Which statement accurately compares the pathophysiology of capture myopathy in translocated wild herbivores to exertional rhabdomyolysis?
(A) Both conditions feature massive skeletal muscle necrosis due to severe metabolic acidosis and ATP depletion.
(B) Capture myopathy solely involves anaerobic glycolysis without lactic acid buildup, whereas exertional rhabdomyolysis is strictly aerobic.
(C) Capture myopathy is characterized by primary renal tubular necrosis independent of myoglobinuria, unlike rhabdomyolysis.
(D) Both conditions resolve spontaneously without systemic consequences once the physical exertion ceases.

2001

22. Which heavy metal bioaccumulation in free-ranging water birds causes severe neurological signs, basophilic stippling in erythrocytes, and characteristic acid-fast intranuclear inclusions in renal tubular epithelial cells?
(A) Mercury
(B) Arsenic
(C) Cadmium
(D) Lead

2015

23. Nipah virus is considered an emerging zoonotic threat at the wildlife-domestic interface. What is the primary reservoir host for this virus?
(A) Feral swine
(B) Pteropid fruit bats
(C) Wild migratory waterfowl
(D) Free-ranging macaques

2008

24. What constitutes the defining histopathological lesion of Myxomatosis in rabbits?
(A) Severe diffuse coagulation necrosis of the hepatic parenchyma
(B) Chronic granulomatous inflammation of the intestinal mucosa
(C) Proliferation of mesenchymal cells in a mucinous matrix
(D) Segmental demyelination of the peripheral nervous system

2018

25. Select the correct sequence of viral pathogenesis events for Rabies virus in a wild canid post-bite exposure.
(A) Centripetal axonal transport → Viral replication in myocytes → Replication in salivary glands → Centrifugal axonal

Downloaded from www.pdf.tube

2019

42. What is the defining feature of specific pathogen-free (SPF) laboratory rodents compared to conventional rodents?

(A) They are known to be free of explicitly defined pathogenic microorganisms.
(B) They have no detectable microorganisms in their bodies.
(C) They are housed outdoors to develop natural immunity to environmental pathogens.
(D) They possess an artificially induced microflora to enhance digestion.

2004

43. Which pathological finding is LEAST relevant to a diagnosis of Feline Panleukopenia in a captive leopard?

(A) Severe segmental necrotizing enteritis
(B) Granulomatous encephalitis
(C) Intestinal crypt epithelial cell necrosis
(D) Severe lymphoid depletion

2006

44. Mammary fibroadenomas are highly prevalent spontaneous neoplasms in aging female Sprague-Dawley rats. What is their typical biological behavior?

(A) They are highly malignant and rapidly metastasize to the lungs.
(B) They are benign, encapsulated masses that can grow to a massive size.
(C) They typically regress spontaneously after the cessation of estrus.
(D) They invade the underlying abdominal musculature deeply without metastasizing.

2023

45. Which of the following best explains why the anticoagulant rodenticide toxicity causes severe hemorrhage in wild carnivores?

(A) It inhibits Vitamin K epoxide reductase, depleting active clotting factors.
(B) It directly destroys circulating blood platelets, leading to profound clinical thrombocytopenia.
(C) It damages the vascular endothelial lining, exposing collagen and depleting fibrinogen.
(D) It irreversibly binds to antithrombin III, causing disseminated intravascular coagulation.

2019

46. What is the primary etiological agent responsible for hepatic coccidiosis in rabbits, leading to severe biliary hyperplasia and cholangitis?

(A) Eimeria perforans
(B) Eimeria magna
(C) Eimeria stiedae
(D) Eimeria intestinalis

2018

47. In zoo and wild birds, Chlamydiosis (Psittacosis) primarily presents with which specific gross pathological lesion?

(A) Severe diphtheritic membranes in the oral cavity and upper esophagus
(B) Severe necrotic enteritis with pseudo-membrane formation
(C) Urate deposition on the visceral surfaces
(D) Fibrinous airsacculitis, hepatomegaly, and splenomegaly

2005

48. What diagnostic feature differentiates Rabbit Hemorrhagic Disease (RHD) from Myxomatosis during a post-mortem examination?

(A) RHD causes purulent rhinitis, whereas Myxomatosis causes hemorrhagic cystitis.
(B) RHD strictly affects the gastrointestinal tract and mucosal linings, whereas Myxomatosis strictly affects the respiratory system.
(C) RHD causes hepatic necrosis and DIC, whereas Myxomatosis causes cutaneous mucinous tumors.
(D) RHD produces basophilic intranuclear inclusions, whereas Myxomatosis produces none.

NODIA

2022

49. Which concept best applies to the pathogenesis of Nutritional Secondary Hyperparathyroidism in captive reptiles?

(A) Excessive dietary calcium levels lead to thyroid C-cell hyperplasia and subsequent hypersecretion of calcitonin.
(B) Deficiency of calcium or Vitamin D3, or an inverted Ca:P ratio, stimulates PTH release and bone resorption.
(C) Primary parathyroid adenomas secrete excessive hormone regardless of dietary intake.
(D) Excessive dietary phosphorus inhibits renal excretion of calcium, leading to hypercalcemia.

2001

50. Given below are two statements: one is asserted as Assertion (A) and the other is labeled as Reason (R).

Assertion (A): Tissue sampling for legal/forensic investigation of a poached elephant must

(D) Hepatic conversion of ammonia to uric acid

2016

34. What is the cause of Inclusion Body Disease (IBD) in captive boid snakes, characterized by neurological signs and regurgitation?
(A) A novel Orthoreovirus
(B) A strain of Ferlavirus
(C) A species-specific Adenovirus
(D) A reptilian Arenavirus

2008

35. The following statements describe the progression of pathological events during capture myopathy in a translocated herbivore:
I. Muscle ischemia and anaerobic glycolysis
II. Extreme physical exertion and sympathetic exhaustion
III. Myoglobinuria and acute renal failure
IV. Lactic acidosis and skeletal muscle necrosis
Which of the following represents the correct sequence of these events?
(A) I → II → IV → III
(B) II → I → IV → III
(C) II → IV → I → III
(D) I → IV → II → III

2024

36. Which of the following is an INCORRECT statement regarding Johne's disease in wild ruminants?
(A) It is caused by the acid-fast bacterium Mycobacterium avium subspecies paratuberculosis.
(B) Gross lesions typically include marked corrugation of the intestinal mucosa.
(C) The disease primarily causes severe caseous pneumonia in the lungs.
(D) Histologically, it is characterized by diffuse granulomatous enteritis.

2011

37. When comparing Mycoplasma pulmonis infection in mice and rats, which pathological feature is predominantly observed in rats rather than mice?
(A) Severe bronchiectasis and cobblestone lungs
(B) Severe unilateral or bilateral purulent otitis media
(C) Acute necrotizing hepatitis with focal microabscesses
(D) Suppurative leptomeningitis with neutrophils in CSF

2011

38. The presence of multinucleated syncytial cells with both intracytoplasmic and intranuclear inclusions in the respiratory epithelium of a wild tiger is most indicative of which infectious agent?
(A) Canine Distemper Virus
(B) Feline Panleukopenia Virus
(C) Feline Calicivirus
(D) Feline Herpesvirus-1

2024

39. Identify the correct statement regarding the clinical presentation and pathology of Simian Immunodeficiency Virus infection in non-human primates.
(A) Macacine herpesvirus 1 - Chronic hepatocellular carcinoma with metastases
(B) Simian Immunodeficiency Virus - $CD4^+$ T lymphocyte depletion and lymphoid atrophy
(C) Mycobacterium tuberculosis - Severe chronic progressive demyelinating peripheral neuropathy
(D) Ebola virus - Severe productive granulomatous pneumonia with hemorrhage

2005

40. Consider the following statements regarding laboratory animal diseases:
I. Sialodacryoadenitis virus causes necrosis of the salivary and lacrimal glands in rats.
II. Demodex musculi is a fungal organism causing dermatophytosis in mice.
III. Corynebacterium kutscheri causes pseudotuberculosis characterized by caseopurulent foci in multiple organs.
IV. Dermatophytosis in rodents is most commonly caused by Trichophyton mentagrophytes.
Which of the above statements is/are INCORRECT?
(A) II
(B) I and IV
(C) II and III
(D) I, III and IV

2014

41. In an investigation of wild herbivore mortality, the discovery of unclotted dark blood oozing from natural orifices and an enlarged, friable spleen strongly suggests infection by which agent?
(A) Pasteurella multocida
(B) Clostridium chauvoei
(C) Mycobacterium bovis
(D) Bacillus anthracis

Click Here to Buy book on Flipkart

191. (B) Adequate adaptation to urea feeding over several weeks
Ruminants can safely consume non-protein nitrogen if given adequate adaptation. Introducing urea slowly over several weeks allows the rumen microflora to adapt, increasing their capacity to utilize ammonia for protein synthesis. This adaptation prevents the rapid accumulation of free ammonia, protecting the animal from hyperammonemia, whereas sudden dietary introduction or low-energy diets promote toxicity.

192. (C) The carcass exhibits widespread, severe pathognomonic inclusion bodies.
Diagnosing non-protein nitrogen toxicity relies on measuring ammonia concentrations in the blood or rumen fluid. Pathologically, excess systemic ammonia inhibits cellular respiration by disrupting the citric acid cycle. The post-mortem autolysis of affected carcasses is exceptionally rapid. However, NPN poisoning does not produce any pathognomonic cellular or viral-like inclusion bodies in tissues.

193. (D) Ammonia
While gross lesions of urea poisoning are highly non-specific and resemble other peracute toxicoses, a characteristic physical sign is detected upon opening the digestive tract. The rumen contents of an animal that died from NPN toxicity typically emit a highly distinct, pungent, and sharp odor of ammonia, which is caused by the massive hydrolysis of urea.

194. (A) Cyclooxygenase (COX)
Non-steroidal anti-inflammatory drugs exert their pharmacological actions and toxic clinical effects primarily by inhibiting the cyclooxygenase enzymes. By blocking both COX-1 and COX-2 isoforms, these medications prevent the conversion of arachidonic acid into active prostaglandins, thromboxanes, and prostacyclins. This inhibition reduces inflammation but also disrupts crucial physiological protective mechanisms in the body.

195. (A) Both A and R are true and R is the correct explanation of A.
NSAID toxicity commonly results in severe gastric erosion and ulceration across multiple animal species. This adverse effect is directly linked to the inhibition of cyclooxygenase-1. By blocking COX-1, NSAIDs deplete protective prostaglandins, specifically PGE_2 and PGI_2. These prostaglandins are essential for maintaining gastric mucosal blood flow, stimulating bicarbonate secretion, and producing protective mucus.

NODIA

196. (B) Renal papillary necrosis
In the kidneys, prostaglandins, particularly PGE_2 and PGI_2, play a vital physiological role by maintaining vasodilation of the afferent arterioles. When NSAIDs inhibit prostaglandin synthesis, renal autoregulation is lost, leading to severe vasoconstriction and localized ischemia. Because the renal medulla has a naturally low blood supply, this ischemic insult leads directly to renal papillary necrosis.

197. (A) Acute visceral gout and renal failure
Diclofenac is an NSAID that exhibits extreme toxicity in avian scavengers, particularly vultures. Ingestion of diclofenac residues from livestock carcasses causes profound inhibition of renal prostaglandins, leading to severe renal ischemia and tubular damage. This results in acute renal failure, which prevents the normal excretion of uric acid, manifesting as widespread visceral gout and sudden death.

198. (C) Gastric erosion and deep ulceration
A dog presenting with melena, hematemesis, and abdominal pain after receiving human ibuprofen is suffering from acute NSAID toxicosis. Human formulations are highly toxic to dogs due to their narrow therapeutic index. The primary lesion is severe gastric erosion and deep mucosal ulceration, which results from the loss of protective prostaglandin-mediated mucosal defense mechanisms.

199. (D) Dehydration and hypovolemia
The nephrotoxic potential of NSAIDs is highly dependent on the physiological state of renal perfusion. Under normal conditions, renal prostaglandins are not highly active. However, during dehydration and hypovolemia, vasoconstrictive hormones are released. Prostaglandins must then induce compensatory vasodilation. Inhibiting them with NSAIDs under these conditions causes unchecked vasoconstriction, severely exacerbating ischemic renal damage.

200. (C) A is true but R is false.
Cats exhibit extreme sensitivity to acetaminophen, rapidly developing methemoglobinemia and

maintain a strict chain of custody.
Reason (R): A strict chain of custody ensures the samples are chemically preserved in 10% neutral buffered formalin without autolysis.
(A) Both (A) and (R) are true and (R) is the correct explanation of (A).
(B) Both (A) and (R) are true but (R) is not the correct explanation of (A).
(C) (A) is true but (R) is false.
(D) (A) is false but (R) is true.

2018
51. An outbreak of Psoroptes cuniculi in a rabbitry will most directly result in which of the following pathological conditions?
(A) Crusty exudative otitis
(B) Severe acute hepatic necrosis
(C) Chronic granulomatous pneumonia
(D) Proliferative enteropathy

2023
52. Newcastle Disease in wild birds can present in a velogenic viscerotropic form. Which anatomical location typically exhibits hemorrhage and necrosis in this specific form?
(A) The cerebral cortex and cerebellar hemispheres
(B) The syrinx, trachea, and primary bronchi
(C) The proventriculus and lymphoid tissue
(D) The renal tubules and ureters

2019
53. Which of the following inferences can be drawn when observing extensive demyelination in the central nervous system of a wild mouse?
(A) The mouse is infected with a neurotropic strain of Murine Hepatitis Virus.
(B) The mouse is likely infected with chronic progressive Murine Mycoplasmosis.
(C) The mouse has consumed a diet deficient in Vitamin C.
(D) The mouse has been exposed to anticoagulant rodenticides.

2011
54. Identify the INCORRECT statement concerning zoonotic diseases encountered during necropsy of laboratory animals.
(A) Macacine herpesvirus 1 causes mild cold sore-like lesions in macaques but fatal disease in humans.
(B) Lymphocytic choriomeningitis virus is a significant hazard when handling infected mouse colonies.
(C) Rabies poses a risk when necropsying wild carnivores with unknown vaccination histories.
(D) Tuberculosis is rarely a concern during necropsy of non-human primates due to natural immunity.

2001
55. Which of the following represents a correct distinction between Hemorrhagic Septicemia and Anthrax?
(A) Hemorrhagic Septicemia causes a chronic wasting disease, whereas Anthrax causes acute necrotizing splenitis.
(B) Hemorrhagic Septicemia is caused by Pasteurella multocida, whereas Anthrax is caused by Bacillus anthracis.
(C) Hemorrhagic Septicemia is transmitted by ticks, whereas Anthrax is transmitted by mosquitoes.
(D) Hemorrhagic Septicemia only affects young animals, whereas Anthrax only affects adults.

2022
56. Which of the following is an expected physiological norm for a healthy, germ-free laboratory rat compared to a conventionally housed rat?
(A) A highly developed and hyperplastic mucosal immune system
(B) Thicker intestinal mucosal walls with significantly increased lamina propria cellularity
(C) An enlarged cecum due to accumulation of undegraded mucins
(D) A significantly higher basal metabolic rate

2017
57. Which viral agent is recognized for causing high mortality in captive elephants through acute hemorrhagic disease and severe myocardial edema?
(A) Lethal systemic Elephantpox virus strain
(B) Foot-and-mouth disease virus
(C) Rabies virus
(D) Elephant Endotheliotropic Herpesvirus

NODIA

2006
58. Hypovitaminosis A in captive psittacines is most commonly characterized by which pathological change?
(A) Squamous metaplasia of the respiratory and salivary epithelium
(B) Diffuse hepatic lipidosis with subsequent pancreatic cell failure and degeneration
(C) Severe rickets and osteomalacia

(D) Highly velogenic neurotropic

2021

77. The presence of eosinophilic intracytoplasmic inclusions (Guarnieri bodies) in epidermal cells is a characteristic finding of which viral infection in laboratory mice?
(A) Ectromelia virus
(B) Murine hepatitis virus
(C) Sendai virus
(D) Sialodacryoadenitis coronavirus

2001

78. What is the fundamental cause of the high mortality observed in Kyasanur Forest Disease among wild primates?
(A) Severe acute fibrinous pneumonia
(B) Granulomatous meningoencephalitis
(C) Hemorrhagic fever causing gastrointestinal bleeding and shock
(D) Profound generalized immunosuppression secondary to systemic lymphoid depletion

2023

79. Which parasitic infestation is most likely responsible for chronic, progressive alopecia and hyperkeratosis in a severely immunocompromised laboratory mouse?
(A) Syphacia obvelata
(B) Aspiculuris tetraptera
(C) Hymenolepis nana
(D) Demodex musculi

2024

80. Consider the following statements about wild animal necropsy:
I. Formalin-fixed tissues are ideal for viral isolation.
II. The necropsy of a suspected anthrax case should proceed with opening the carcass thoroughly.
III. Heart blood smears are useful for diagnosing anthrax in free-ranging herbivores.
IV. Legal forensics requires maintaining a chain of custody for all samples.
Which of the above statement(s) is/are correct?
(A) I and II
(B) III and IV
(C) I, III and IV
(D) II, III and IV

2014

81. In rabbits, what is the most appropriate anatomical site to sample when investigating a suspected infection with Psoroptes cuniculi?
(A) The interdigital spaces
(B) The external ear canal
(C) The conjunctival sac
(D) The perianal region

2002

82. Which statement accurately describes the etiopathology of Myxomatosis in wild and domestic rabbits?
(A) It is a Calicivirus that causes massive hepatic necrosis.
(B) It is a severe bacterial disease causing bilateral purulent conjunctivitis and rhinitis.
(C) It primarily affects the central nervous system, causing paralysis.
(D) It is caused by a Leporipoxvirus, transmitted by fleas and mosquitoes.

2006

83. Identify the INCORRECT pair regarding nutritional and metabolic disorders.
(A) Gout in reptiles - Excessive retention of urea
(B) Vitamin C deficiency in guinea pigs - Defective collagen synthesis
(C) Pregnancy toxemia in guinea pigs - Ketosis and fatty liver
(D) Hypovitaminosis A in birds - Squamous metaplasia

2010

84. Which viral agent causes a persistent infection in macaques that closely mimics the pathogenesis of human HIV, leading to opportunistic infections?
(A) Macacine herpesvirus 1
(B) Simian hemorrhagic fever virus
(C) Simian Immunodeficiency Virus
(D) Measles virus

2011

85. Organophosphate poisoning in free-ranging wildlife exerts its toxic effect primarily by:
(A) Inhibiting acetylcholinesterase, causing overstimulation of cholinergic receptors
(B) Enhancing the release of gamma-aminobutyric acid (GABA)
(C) Irreversibly binding to hemoglobin molecules, completely preventing oxygen transport
(D) Uncoupling oxidative phosphorylation in the mitochondria

2006

86. Which lesion is most characteristic of chronic progressive nephropathy (CPN) in aging laboratory rats?
(A) Severe suppurative tubulointerstitial nephritis with bacterial colonies
(B) Massive renal cortical necrosis

Downloaded from www.pdf.tube

(D) They can cause severe respiratory and enteric diseases in aberrant hosts.

2001

68. Which gross lesion is most appropriately associated with an acute outbreak of Foot-and-mouth disease in wild ungulates?
(A) Caseous granulomas primarily in the retropharyngeal lymph nodes
(B) Massive hepatic necrosis and gallbladder edema
(C) Vesicular lesions on the oral mucosa and interdigital clefts
(D) Severe fibrinous pleuropneumonia

2013

69. A toxicological investigation of a free-ranging vulture reveals massive uric acid deposition on the heart and liver. This is directly linked to the ingestion of which veterinary drug in the food chain?
(A) Diclofenac
(B) Meloxicam
(C) Ivermectin
(D) Oxytetracycline

2008

70. Consider the following statements regarding viral diseases of laboratory mice:
I. Murine hepatitis virus belongs to the family Coronaviridae.
II. Sendai virus infection promotes secondary bacterial pneumonias.
III. Mousepox virus is highly contagious and fatal in all known mouse strains.
Which of the above statement(s) is/are correct?
(A) I and II
(B) I
(C) II and III
(D) I, II and III

2021

71. The pathology of lead poisoning in waterfowl frequently includes all of the following EXCEPT:
(A) Impaction of the proventriculus and gizzard
(B) Severe vesicular glossitis
(C) Acid-fast intranuclear inclusions in renal tubular epithelium
(D) Demyelination of peripheral nerves leading to paresis

2025

72. Which of the following is considered the primary biosafety protocol when performing a necropsy on a non-human primate suspected of Macacine herpesvirus 1 infection?
(A) Performing the complete necropsy outdoors under direct sunlight to allow for UV light sterilization
(B) Wearing standard surgical scrubs and a simple surgical mask
(C) Utilizing a biosafety cabinet and wearing full personal protective equipment
(D) Washing the cadaver with 10% formalin before initiating the necropsy

2024

73. Identify the correct statement regarding dermatophytosis in laboratory rodents.
(A) It is primarily caused by Demodex musculi.
(B) It consistently causes intense localized pruritus leading to severe self-mutilation.
(C) The lesions are strictly confined to the ventral abdomen.
(D) Trichophyton mentagrophytes is a common etiology with zoonotic potential.

NODIA

2008

74. Which pathological feature provides the most accurate basis for classifying a lesion as amyloidosis in aging laboratory rodents?
(A) Extracellular eosinophilic material that stains with Congo Red showing green birefringence
(B) The presence of lipofuscin within hepatocytes
(C) The intracellular accumulation of numerous proteinaceous hyaline droplets within renal proximal tubules
(D) The proliferation of fibrous connective tissue in the renal interstitium

2022

75. A group of translocated deer exhibit severe muscle stiffness, ataxia, and dark brown urine a day after capture. What is the definitive pathophysiological cause of the urine color?
(A) Hemoglobinuria secondary to acute intravascular hemolysis
(B) Myoglobinuria from exertional rhabdomyolysis
(C) Hematuria from renal trauma
(D) Bilirubinuria from acute hepatic failure

2017

76. In captive wild birds, Newcastle disease virus is classified into pathotypes based on virulence. Which pathotype is responsible for severe hemorrhagic lesions in the gastrointestinal tract?
(A) Lentogenic
(B) Velogenic viscerotropic
(C) Mesogenic

(A) Eimeria stiedae
(B) Pasteurella multocida
(C) Myxoma virus
(D) Eimeria intestinalis

2017

126. A toxicological investigation in a captive zoo bird reveals marked thinning of eggshells and reproductive failure. Which environmental toxicant is classically associated with this pathology?
(A) Organophosphate pesticides
(B) Anticoagulant rodenticides
(C) DDT and its metabolites
(D) Lead shot contamination

2003

127. Which of the following is an expected physiological finding during the necropsy of a young, healthy guinea pig, which should NOT be confused with pathology?
(A) Severe, generalized enlargement of the systemic lymph nodes
(B) A large, thin-walled cecum occupying much of the abdomen
(C) Severe, diffuse alopecia extending across the entire dorsum
(D) Multiple petechial hemorrhages visible on both kidney cortices

2014

128. What distinguishes the macroscopic appearance of Tyzzer's disease in the liver of a mouse from that of Salmonella infection?
(A) Tyzzer's disease presents as large, single, caseous abscesses, while Salmonella infection presents as widespread diffuse hepatic fibrosis.
(B) Tyzzer's disease causes a massively enlarged, green-tinged liver, while Salmonella causes a shrunken, severely fibrotic hepatic parenchyma.
(C) There is absolutely no visible macroscopic difference between the two diseases; they are completely and grossly identical at necropsy.
(D) Tyzzer's causes multiple pale, miliary necrotic foci, while Salmonella causes hepatosplenomegaly with focal histiocytic nodules.

2008

129. Which of the following describes the most relevant histopathological lesion for confirming a diagnosis of Peste des Petits Ruminants in a wild goat?
(A) Syncytial cells with eosinophilic intracytoplasmic and intranuclear inclusions in respiratory and intestinal epithelia.
(B) Diffuse granulomatous enteritis with multinucleated giant cells containing abundant acid-fast bacillary organisms.
(C) Massive hepatocellular necrosis characterized by large, prominent basophilic intranuclear inclusion bodies.
(D) Widespread, bilateral demyelination of the white matter tracts located throughout the spinal cord parenchyma.

2025

130. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R).
Assertion (A): Zoonotic disease transmission is a negligible risk when performing a necropsy on a captive reptile.
Reason (R): Reptiles are frequent asymptomatic carriers of zoonotic Salmonella species that pose a serious infection risk to humans.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2012

131. Identify the correct pair associating the specific laboratory animal pathogen with its primary diagnostic testing modality for colony surveillance.
(A) Murine Mycoplasmosis - Serology and PCR
(B) Syphacia obvelata - Standard serology ELISA
(C) Corynebacterium kutscheri - Fecal flotation
(D) Demodex musculi - Routine blood culture

2013

132. What is the cause of death in wild carnivores affected by the paralyzing tick-borne disease, assuming it progresses to its final stage without intervention?
(A) Severe acute hepatic failure and encephalopathy
(B) Massive internal hemorrhage into body cavities
(C) Acute renal tubular necrosis and severe uremia
(D) Respiratory failure from ascending paralysis

Click Here to Buy book on Amazon

(C) Multiple renal cell carcinomas
(D) Thickened glomerular basement membranes and protein casts

2025

87. An adult captive chimpanzee dies suddenly. Necropsy reveals extensive granulomas in the lungs and bronchial lymph nodes. Acid-fast bacilli are identified in smears. What is the most significant occupational hazard consequence for the veterinary staff?
(A) Significant risk of exposure to a highly contagious zoonotic systemic mycotic infection
(B) Risk of exposure to Mycobacterium tuberculosis, requiring immediate staff screening
(C) Minimal risk, as this pathogen is strictly host-specific to apes
(D) Risk of developing severe systemic allergic reactions

2012

88. What is the primary cause of death in domestic rabbits infected with Rabbit Hemorrhagic Disease Virus (RHDV)?
(A) Severe acute dehydration secondary to profuse watery or hemorrhagic diarrhea
(B) Asphyxiation due to fibrinous pleuropneumonia
(C) Acute hepatic necrosis leading to DIC and hemorrhage
(D) Status epilepticus caused by acute encephalitis

2018

89. Which of the following describes the most relevant gross finding of Avian Influenza in susceptible wild bird populations?
(A) Prolific, crusted, wart-like lesions on the unfeathered parts of the skin
(B) Caseous nodules within the crop and esophagus
(C) Unilateral enlargement of the sciatic nerve
(D) Cyanosis of the comb and wattles, with petechiae on visceral organs

2001

90. Given below are two statements: one is asserted as Assertion (A) and the other is labeled as Reason (R).
Assertion (A): Specific Pathogen Free (SPF) laboratory animals are not necessarily germ-free.
Reason (R): SPF animals have been demonstrated to be free of specific named pathogens but may harbor a normal commensal microflora.
(A) Both (A) and (R) are true and (R) is the correct explanation of (A).
(B) Both (A) and (R) are true but (R) is not the correct explanation of (A).
(C) (A) is true but (R) is false.
(D) (A) is false but (R) is true.

2012

91. Identify the INCORRECT statement regarding Sialodacryoadenitis virus (SDAV) infection in rats.
(A) It is caused by a highly contagious coronavirus.
(B) It results in a severe, highly fatal gastroenteritis in adult rats.
(C) It causes inflammation and necrosis of the salivary and lacrimal glands.
(D) Clinical signs often include cervical edema and porphyrin staining around the eyes.

NODIA

2008

92. Which pinworm species of laboratory mice is diagnosed primarily by fecal flotation or examination of colonic contents for symmetrical, ellipsoidal ova, because the adult females do not migrate to the perianal region to deposit eggs?
(A) Syphacia obvelata
(B) Aspiculuris tetraptera
(C) Trichosomoides crassicauda
(D) Rodentolepis nana

2013

93. What is the most appropriate action if a carcass of a wild elephant is suspected of having died from Anthrax?
(A) Do not open the carcass; take a peripheral blood smear and inform authorities.
(B) Perform a full detailed necropsy immediately to evaluate all internal organs for lesions.
(C) Transport the entire carcass to a central laboratory for detailed dissection.
(D) Burn the carcass immediately without taking any diagnostic samples.

2004

94. Which of the following pathological changes is typically associated with heavy metal bioaccumulation (specifically Cadmium) in wildlife?
(A) Severe demyelination of the central nervous system pathways
(B) Granulomatous pneumonia
(C) Renal tubular necrosis and osteomalacia
(D) Proliferative dermatitis

articular cartilage.
(C) A persistent viral infection of the synovial membranes that triggers massive localized inflammation.
(D) Defective purine metabolism or renal impairment leading to hyperuricemia and joint deposition.

2021
110. Consider the following statements about laboratory rabbit diseases:
I. Pasteurella multocida can cause otitis media and interna, leading to torticollis (wry neck).
II. Rabbit hemorrhagic disease virus targets hepatocytes, causing acute hepatic necrosis.
III. Myxoma virus causes severe hemorrhagic enteritis in domestic rabbits.
Which of the above statement(s) is/are INCORRECT?
(A) I and II
(B) III
(C) I and III
(D) II

2015
111. What is the most relevant consequence of a coronavirus spillover event from a bat reservoir to an intermediate wild animal host (like a civet)?
(A) The virus rapidly kills the intermediate host, preventing any further transmission to other species.
(B) The intermediate host allows replication and mutation, facilitating adaptation for human infection.
(C) The virus permanently reverts to a completely non-pathogenic state in the intermediate host reservoir.
(D) The intermediate host produces neutralizing antibodies that provide passive immunity to local humans.

2018
112. Identify the correct pair of viral disease and its prominent histopathological feature in birds.
(A) Avian Influenza - Eosinophilic intracytoplasmic inclusions in tracheal epithelium
(B) Psittacosis - Basophilic intranuclear inclusions in hepatocytes
(C) Marek's disease - Fibrous osteodystrophy
(D) Newcastle Disease - Non-suppurative encephalomyelitis and lymphoid necrosis

2009
113. In a laboratory colony of nude (athymic) mice, which infectious agent causes a chronic, severe, wasting disease primarily due to the lack of T-cell mediated immunity, whereas it causes only a self-limiting infection in immunocompetent mice?
(A) Mouse hepatitis virus
(B) Mousepox virus
(C) Corynebacterium kutscheri
(D) Salmonella Typhimurium

2005
114. Which finding is LEAST expected during the necropsy of a laboratory rat suffering from chronic, severe Mycoplasma pulmonis infection?
(A) Suppurative rhinitis
(B) Bronchiectasis with purulent exudate
(C) Eosinophilic meningoencephalitis
(D) Fibrinopurulent otitis media

2019
115. A toxicological examination of a poisoned wild dog reveals bright red mucous membranes, cherry-red blood, and an odor of bitter almonds from the stomach contents. Which toxicant is the cause?
(A) Cyanide
(B) Organophosphates
(C) Anticoagulant rodenticides
(D) Lead

2014
116. How does the pathological presentation of Dirofilaria immitis infection in wild canids primarily differ from its presentation in domestic felines?
(A) Canids harbor the adult parasites strictly in the GI tract, whereas felines harbor them exclusively within the heart.
(B) The parasite causes acute hepatic necrosis in wild canids, while causing only a mild localized dermatitis in felines.
(C) There are absolutely no pathological or clinical differences between the two animal groups; the disease is identical.
(D) Canids typically develop heavy worm burdens, while felines exhibit severe respiratory signs from very few worms.

2022
117. What definition best fits "Inclusion Body Disease" (IBD) in snakes?
(A) A chronic bacterial infection causing caseous pulmonary granulomas with prominent bacterial colonies mimicking inclusions.
(B) An arenavirus infection of boid

Downloaded from www.pdf.tube

2004

102. What differentiates the pathology of Hemorrhagic Septicemia (HS) from other pneumonic conditions in wild ruminants?

(A) HS strictly causes chronic, caseous necrotic nodules in the lung lobes without any systemic or vascular involvement.
(B) HS is characterized by marked corrugation of the intestinal mucosa and chronic wasting.
(C) HS presents exclusively as a neurological disease with microabscesses in the brainstem.
(D) HS presents with edematous swelling of the head, neck, brisket, and petechial hemorrhages.

2023

103. In evaluating the pathology of a free-ranging bird that died from suspected anticoagulant rodenticide toxicity, which of the following is the most likely gross lesion?

(A) Severe impaction of the gizzard with massive amounts of undigested fibrous material
(B) Extensive hemocoelom and hematomas
(C) Urate deposits over the epicardium and liver
(D) Fibrinous airsacculitis

2015

104. Which of the following combinations of gross lesions is most characteristic of fatal Canine Distemper Virus infection in wild canids?

(A) Massive hepatic necrosis and hemorrhagic cystitis
(B) Vegetative endocarditis and renal infarcts
(C) Suppurative bronchopneumonia, catarrhal enteritis, and enamel hypoplasia
(D) Severe acute hemorrhagic enteritis with complete preservation of the entire respiratory tract

2005

105. The following statements describe the events in the pathogenesis of Nutritional Secondary Hyperparathyroidism (NSHP) in a captive reptile:

I. Parathyroid hormone (PTH) release is stimulated
II. Dietary deficiency of calcium or inversion of Ca:P ratio occurs
III. Fibrous tissue replaces resorbed bone (Fibrous osteodystrophy)
IV. Calcium is mobilized from skeletal stores leading to bone weakening

Which of the following represents the correct sequence of these events?

(A) II → I → IV → III
(B) I → II → III → IV
(C) II → IV → I → III
(D) I → III → II → IV

NODIA

2012

106. Which is the most appropriate diagnostic test to confirm an active Nipah virus infection in a wildlife sample during a spillover investigation?

(A) Bacterial culture of lung tissue on sheep blood agar
(B) Real-time PCR of oropharyngeal swabs or target tissues
(C) Fecal flotation and sedimentation for parasitic ova
(D) Direct immunofluorescence of superficial skin biopsies

2023

107. Which of the following is an INCORRECT statement regarding rabies in wild carnivores?

(A) The virus travels to the central nervous system via hematogenous spread.
(B) The virus produces pathognomonic eosinophilic intracytoplasmic inclusions called Negri bodies.
(C) Clinical signs typically include behavioral changes, aggression, and paralysis.
(D) Definitive diagnosis usually requires fluorescent antibody testing of brain tissue.

2018

108. A captive rhino presents with acute death, blood exuding from orifices, and an incomplete rigor mortis. What is the fundamental biosafety implication for the pathology team?

(A) The team must wear specialized lead aprons to prevent heavy metal exposure during the dissection.
(B) The team must immediately perform a field necropsy to evaluate the gastrointestinal tract and organs.
(C) The team must halt the necropsy and handle the carcass as a suspected Bacillus anthracis case.
(D) There are no specific biosafety concerns, and the routine field necropsy can safely proceed.

2007

109. What physiological abnormality primarily drives the pathogenesis of articular gout in birds?

(A) An autoimmune response targeting synovial fluid proteins and causing severe local joint erosion.
(B) Excessive dietary protein intake causing direct toxic damage and degeneration of

(D) Severe bilateral demyelination of the spinocerebellar tracts

2025

59. When eliminating potential causes of high mortality with concurrent diarrhea in a rat colony, which agent is LEAST likely to be responsible?
(A) Murine Hepatitis Virus
(B) Syphacia obvelata
(C) Salmonella enterica
(D) Clostridium piliforme

2002

60. Match the rodent infectious agents in List I with their respective disease names/lesions in List II.
List I: (Infectious Agents)
P. Corynebacterium kutscheri
Q. Sialodacryoadenitis virus
R. Aspiculuris tetraptera
S. Trichophyton mentagrophytes
List II: (Diseases/Lesions)
1. Cervical swelling and porphyrin staining
2. Ringworm/Dermatophytosis
3. Pinworm infection
4. Murine pseudotuberculosis
(A) P-4, Q-1, R-3, S-2
(B) P-4, Q-3, R-1, S-2
(C) P-1, Q-4, R-2, S-3
(D) P-2, Q-1, R-4, S-3

2006

61. Identify the correct statement concerning Tuberculosis in wild non-human primates.
(A) Infection results in classic caseating granulomas in the lungs.
(B) It predominantly presents as an acute hemorrhagic septicemia.
(C) Lesions are typically strictly confined to the mucosa of the gastrointestinal tract.
(D) The disease is non-zoonotic and poses no risk to animal handlers.

2008

62. Which of the following processes represents the initial cellular pathogenesis of Canine Distemper Virus in a susceptible wild canid?
(A) Direct active invasion of the central nervous system pathways via the olfactory nerve fibers
(B) Replication in the intestinal crypt cells leading to complete villous atrophy
(C) Respiratory entry, local lymphoid replication, and systemic dissemination
(D) Infection of erythrocytes causing intravascular hemolysis

2024

63. Which of the following is a classic pathological finding associated with Helicobacter hepaticus infection in susceptible strains of laboratory mice (e.g., A/J mice)?
(A) Acute hemorrhagic enteritis and intestinal mucosal sloughing
(B) Chronic active hepatitis and hepatic portal fibroplasia with biliary hyperplasia
(C) Severe unilateral purulent otitis media and head tilt
(D) Necrotic dermatitis of the tail and extremities

2009

64. A wild feline presents with severe, acute anemia, hemoglobinuria, and icterus. Blood smears reveal intraerythrocytic pyriform bodies. What is the most likely etiology?
(A) Trypanosomiasis
(B) Dirofilariasis
(C) Babesiosis
(D) Anthrax

2011

65. Which parasite of laboratory rats resides in the urinary bladder and can cause epithelial hyperplasia and predisposition to tumor development?
(A) Trichosomoides crassicauda
(B) Capillaria hepatica
(C) Syphacia muris
(D) Hymenolepis nana

2008

66. How does the pathology of Trypanosoma evansi (Surra) typically manifest in wild carnivores like tigers?
(A) Severe hemorrhagic gastroenteritis with extensive mucosal ulceration
(B) Multiple cutaneous granulomas
(C) Diffuse interstitial pneumonia
(D) Petechial hemorrhages, edema, and meningoencephalitis

2010

67. Which of the following statements about Coronaviruses at the wildlife-domestic interface is INCORRECT?
(A) They typically cause high mortality in their natural bat reservoir hosts.
(B) Bats are considered significant natural reservoirs for many emerging coronaviruses.
(C) Spillover events often involve intermediate hosts such as civets or pangolins.

Click Here to Buy book on Flipkart

snakes characterized by eosinophilic intracytoplasmic inclusions in CNS and other tissues.
(C) A metabolic disorder caused by severe calcium deficiency leading to the formation of crystalline kidney inclusions.
(D) A chronic protozoal infection of the circulatory system characterized by numerous pigmented intraerythrocytic parasites.

2015
118. Which statement regarding the pathology of Elephant Endotheliotropic Herpesvirus (EEHV) is correct?
(A) It strictly affects older, captive elephants, causing chronic wasting and lethargy.
(B) It results in massive skin tumors that slowly obscure the host's vision and feeding.
(C) It causes acute, highly fatal hemorrhagic disease in juvenile Asian elephants.
(D) It is a respiratory pathogen causing severe, caseating granulomatous lung lesions.

2003
119. What is the fundamental cause of death in wild birds poisoned by diclofenac?
(A) Massive acute hepatic necrosis
(B) Severe respiratory depression and asphyxiation
(C) Profound immunosuppression and secondary septicemia
(D) Acute renal failure leading to visceral gout

2001
120. Consider the following statements about infectious diseases in wild felines:
I. Feline panleukopenia virus targets rapidly dividing cells in the bone marrow and intestinal crypts.
II. Canine distemper virus can infect large wild felids, causing fatal neurological and respiratory disease.
III. Trypanosoma evansi causes a severe, fatal demyelinating encephalomyelitis in tigers.
IV. Rabies virus strictly causes the "dumb" form of the disease in wild felids.
Which of the above statement(s) is/are correct?
(A) I and II
(B) III and IV
(C) I, II and IV
(D) II, III and IV

2024
121. In specific pathogen-free (SPF) laboratory rodents, the incidental finding of amyloidosis at necropsy is most closely correlated with which factor?
(A) High levels of dietary vitamin C supplementation
(B) Advancing age and genetic strain
(C) Acute viral infection exposure
(D) Chronic exposure to heavy metals

2011
122. Which process is directly responsible for the massive, firm enlargement of the mammary glands observed in female rats with mammary fibroadenoma?
(A) Malignant proliferation of epithelial cells invading local lymph nodes.
(B) Benign proliferation of glandular epithelium and fibrous stroma.
(C) Severe purulent mastitis resulting from acute Staphylococcus infection.
(D) Accumulation of caseous necrotic material within the lactiferous ducts.

2003
123. Identify the INCORRECT statement regarding the pathology of pasteurellosis in rabbits.
(A) It causes acute, massive hepatic necrosis as its primary lesion.
(B) It frequently presents as a mucopurulent rhinitis.
(C) It can spread hematogenously to cause abscesses in multiple organs.
(D) It can cause fibrinous pneumonia and pleuritis.

2004
124. What is the primary advantage of gnotobiotic animals in the study of infectious diseases?
(A) Their highly developed immune systems mimic complex human physiological responses with absolute perfection.
(B) They possess an inherent genetic resistance to virtually all classes of common experimental pathogens.
(C) Their defined microflora allows controlled studies of host-pathogen interactions without interference.
(D) They require standard animal facilities without any specialized housing or sterile barrier equipment.

2011
125. The presence of raised, white, multifocal lesions on the serosal surface of the intestines of a rabbit is strongly suggestive of infection by which organism?

Downloaded from www.pdf.tube

2004

95. The pathogenesis of pregnancy toxemia in guinea pigs is driven by a primary metabolic disturbance. Which of the following triggers the cascade leading to hepatic lipidosis and death?

(A) Excessive carbohydrate intake during late gestation
(B) Severe dietary calcium deficiency or phosphorus imbalance leading to hypocalcemia
(C) Viral infection of the pancreatic islets
(D) Negative energy balance leading to fat mobilization and ketogenesis

2015

96. Which inference can be made about a captive macaque that tests positive for Macacine herpesvirus 1 but shows no clinical signs?

(A) The macaque has cleared the virus and is no longer infectious.
(B) The test is definitively a false positive, as the virus is always fatal in macaques.
(C) The macaque is a latent carrier and poses a zoonotic risk to human handlers when stressed.
(D) The macaque requires immediate emergency euthanasia as it will develop fatal encephalitis within several days.

2009

97. In a laboratory rodent facility, which management practice is LEAST relevant to controlling an outbreak of Murine Mycoplasmosis?

(A) Maintaining a strictly defined SPF barrier system
(B) Treating infected animals to eliminate the carrier state.
(C) Reducing environmental ammonia levels in the animal rooms
(D) Implementing highly strict quarantine protocols and routine diagnostic testing of incoming animals.

2005

98. An outbreak of hemorrhagic enteritis and acute death in a colony of germ-free mice after accidental exposure to a conventional environment is an example of:

(A) Competitive exclusion by normal flora
(B) Enhanced immune competence of germ-free animals
(C) Spontaneous genetic mutation causing sudden immune deficiency in the mouse strain
(D) The high susceptibility of germ-free animals to common environmental pathogens

NODIA

2012

99. Which of the following pathological findings is highly characteristic of Ranavirus infection in susceptible captive amphibians and turtles?

(A) Progressive, chronic dry gangrene of the digits and tail
(B) Systemic hemorrhages, multifocal splenic and hepatic necrosis, and cutaneous ulceration
(C) Severe proliferative osteodystrophy of long bones
(D) Extensive demyelination of the central nervous system

2010

100. Match the diseases in List I with their characteristic pathological findings in List II.

List I: (Diseases)

P. Feline Panleukopenia
Q. Elephant Endotheliotropic Herpesvirus
R. Johne's Disease
S. Peste des Petits Ruminants

List II: (Pathological Findings)

1. Erosive stomatitis and bronchointerstitial pneumonia
2. Intestinal crypt necrosis and lymphoid depletion
3. Myocardial edema and capillary endothelial inclusions
4. Granulomatous enteritis with acid-fast bacilli

(A) P-1, Q-3, R-2, S-4
(B) P-2, Q-3, R-4, S-1
(C) P-2, Q-4, R-3, S-1
(D) P-3, Q-2, R-1, S-4

2015

101. Which definition accurately describes "capture myopathy" in wild herbivores?

(A) An acute non-infectious muscle disease triggered by extreme stress of pursuit and capture.
(B) A primary hereditary muscle disease leading to spontaneous, progressive breakdown of muscle fibers during rest.
(C) An infectious myositis caused by Clostridium chauvoei introduced through capture wounds.
(D) A nutritional deficiency of Vitamin E and Selenium exacerbated by the capture process.

2004

133. Which of the following inferences is most accurate regarding a rhesus macaque demonstrating vesicles and ulcers on the tongue and lips?
(A) The animal is suffering from an active, acute Foot-and-mouth disease infection.
(B) The animal likely has Macacine herpesvirus 1 and requires extreme biosafety.
(C) The animal has developed clinical scurvy secondary to chronic Vitamin C deficiency.
(D) The animal is exhibiting clinical signs of advanced Simian Immunodeficiency Virus.

2015

134. Which condition represents the most severe complication of untreated infectious stomatitis (mouth rot) in captive snakes?
(A) Transformation into squamous cell carcinoma
(B) Development of severe inclusion body disease
(C) Osteomyelitis and systemic septicemia
(D) Ascending motor paralysis of the tail

2011

135. The presence of a severe, acute, fibrinohemorrhagic pleuropneumonia in a young wild bighorn sheep is most strongly indicative of which primary bacterial infection?
(A) Mycobacterium avium paratuberculosis
(B) Clostridium chauvoei
(C) Brucella abortus
(D) Pasteurella or Mannheimia species

2009

136. Which of the following eliminates nutritional secondary hyperparathyroidism as a primary diagnosis in a captive iguana presenting with bone fractures?
(A) A history of consuming calcium-dusted, UV-exposed diet
(B) The presence of severely pliable, soft mandibles (rubber jaw)
(C) Radiographic evidence showing profoundly decreased bone density
(D) Routine blood biochemistry revealing severe systemic hypocalcemia

2016

137. Which statement accurately describes the etiopathology of Kyasanur Forest Disease (KFD)?
(A) KFD is a paramyxovirus causing severe respiratory distress and systemic failure in macaques.
(B) KFD is a tick-borne flavivirus causing hemorrhagic fever in humans and mortality in monkeys.
(C) KFD is a herpesvirus causing chronic latent infections within wild boar populations.
(D) KFD is a filovirus primarily harbored by fruit bats that leads to acute systemic disease.

2016

138. In the pathology of specific pathogen-free animals, what is the consequence if the barrier system fails and mice are exposed to Murine Norovirus (MNV)?
(A) The colony will experience immediate, massive mortality due to acute necrotizing encephalitis.
(B) Most immunocompetent mice remain asymptomatic, but the virus can confound immunological research.
(C) The mice will develop severe, purulent conjunctivitis and rhinitis with systemic distress.
(D) All mice will rapidly develop aggressive mammary tumors throughout their subcutaneous tissue.

2013

139. What is the fundamental distinction in the pathology of acute vs. chronic lead poisoning in waterfowl?
(A) Acute poisoning causes rapid death with proventricular distension, while chronic causes emaciation and gall bladder distension.
(B) Acute poisoning strictly causes severe neurological signs, whereas chronic lead exposure solely results in multiple bone fractures.
(C) Acute poisoning causes massive and rapid hepatic failure, whereas chronic lead exposure leads directly to primary respiratory failure.
(D) There are absolutely no clinical or pathological distinctions; both acute and chronic presentations yield identical gross lesions.

2009

140. Match the viral diseases of wildlife with their respective viral families.

List I: (Viral Disease)
P. Nipah virus
Q. Kyasanur Forest Disease virus
R. Canine Distemper virus
S. Elephant Endotheliotropic Herpesvirus

List II: (Viral Family)
1. Flaviviridae
2. Paramyxoviridae (Henipavirus)
3. Herpesviridae

Click Here to Buy book on Amazon

(B) AI viruses are classified strictly based on their specific H and N surface glycoproteins.
(C) The virus can be readily transmitted through both respiratory secretions and infected feces.
(D) Lesions of highly pathogenic AI can include edema, hemorrhage, and necrosis in multiple organs.

2018
188. Which is the most appropriate technique for preserving feces intended for parasitological examination from a free-ranging wild animal?
(A) Placing the collected feces in a 10% neutral buffered formalin solution.
(B) Freezing the fecal specimens immediately inside a deep freezer at -80°C.
(C) Mixing feces with 10% polyvinyl alcohol or refrigerating fresh samples.
(D) Placing the collected fecal specimens in a 70% ethanol solution.

2005
189. The presence of multifocal, white, caseous nodules on the epicardium, liver, and air sacs of a captive raptor, combined with a history of a high-protein diet and dehydration, is strongly indicative of:
(A) Avian Tuberculosis
(B) Aspergillosis
(C) Chlamydiosis
(D) Visceral gout

2010
190. Consider the following statements about zoonoses:
I. Nipah virus causes severe encephalitis in humans.
II. Macacine herpesvirus 1 is fatal in humans but causes mild disease in macaques.
III. Rabies is uniformly fatal in humans once clinical signs appear.
IV. Avian influenza H5N1 cannot infect humans under any circumstances.
V. Tuberculosis in non-human primates poses no risk to humans.
Which of the above statement(s) is/are correct?
(A) I, II and III
(B) I, IV and V
(C) II, III, IV and V
(D) I, II, III and V

NODIA

2013
191. Which pathological finding differentiates infectious stomatitis from inclusion body disease in a captive snake?
(A) Infectious stomatitis strictly causes bone fractures of the jaw, whereas inclusion body disease causes chronic oral ulcers.
(B) Stomatitis causes oral petechiae, caseous exudate, and ulcers, while inclusion body disease causes neurological signs and inclusions.
(C) Infectious stomatitis is a primary viral infection, whereas systemic inclusion body disease is caused by bacterial pathobionts.
(D) There is absolutely no clinical or pathological difference between them; both conditions are completely synonymous terms.

2012
192. What is the fundamental physiological reason that exertional rhabdomyolysis causes acute renal failure in translocated wild herbivores?
(A) The physical capture process directly causes severe, acute mechanical trauma to the kidney structures.
(B) The intense stress response releases massive levels of cortisol, which directly destroys glomeruli.
(C) Dehydration causes the blood to become severely hyperviscous, leading to multiple renal infarctions.
(D) Muscle necrosis releases myoglobin into the blood, which precipitates in and damages renal tubules.

2007
193. A free-ranging vulture is found dead with severe visceral gout. Which toxicant, commonly used as a non-steroidal anti-inflammatory drug (NSAID) in domestic livestock, is the established cause of this syndrome?
(A) Diclofenac
(B) Flunixin meglumine
(C) Carprofen
(D) Meloxicam

2018
194. Which of the following is an expected histopathological lesion in a wild felid suffering from feline panleukopenia?
(A) Proliferation of the intestinal crypt epithelium forming adenomas.
(B) Hepatocellular necrosis with large basophilic intranuclear inclusions.
(C) Depletion of lymphoid tissue and necrosis of

a solution of 70% ethanol.
(C) Drying the specimens completely inside a biological safety cabinet.
(D) Placing tissues in 10% neutral buffered formalin at a 10:1 ratio.

2010

149. The presence of multifocal caseous nodules in the lungs, liver, and spleen of a captive macaque, which stain positive with Ziehl-Neelsen stain, definitively indicates infection with:
(A) Simian Immunodeficiency Virus
(B) Mycobacterium tuberculosis
(C) Coccidioides immitis fungus
(D) Corynebacterium species

2001

150. Consider the following statements about zoonoses at the wildlife-domestic interface:
I. Nipah virus is transmitted via contact with secretions from infected Pteropus bats.
II. Severe Acute Respiratory Syndrome (SARS) coronaviruses utilize wild animals as intermediate hosts.
III. Kyasanur Forest Disease is a tick-borne viral hemorrhagic fever.
IV. Rabies virus only infects wild carnivores, sparing domestic herbivores.
V. Avian influenza H5N1 can spill over into domestic poultry and humans.
Which of the above statement(s) is/are correct?
(A) I, II, III and V
(B) I, II and III
(C) I, III, IV and V
(D) II, III and V

2006

151. Which pathological finding differentiates Tyzzer's disease from Salmonella infection in laboratory rodents?
(A) Salmonella produces large, multinucleated giant cells in the liver, whereas Tyzzer's disease never forms giant cells.
(B) Tyzzer's disease only affects the upper respiratory tract, leaving the liver tissue entirely free of pathology.
(C) Salmonella infection in these rodents exclusively causes severe, chronic exudative dermatitis and alopecia.
(D) Tyzzer's features intracellular silver-staining bacilli in parallel bundles within hepatocytes near necrotic margins.

2022

152. What is the fundamental physiological reason that guinea pigs are highly susceptible to scurvy?
(A) They lack L-gulonolactone oxidase, making them unable to synthesize ascorbic acid from glucose.
(B) They have a high metabolic requirement for Vitamin C that cannot be met by standard dietary intake alone.
(C) Their gastrointestinal tract rapidly degrades dietary Vitamin C before any physiological absorption occurs.
(D) They possess a genetic defect that prevents Vitamin C from crossing target cell membranes.

2009

153. A free-ranging tiger is found dead with frothy, blood-tinged fluid exuding from the nostrils. Necropsy reveals severe pulmonary edema and an enlarged, soft spleen. Blood smears reveal large, square-ended, Gram-positive bacilli. What is the diagnosis?
(A) Hemorrhagic septicemia
(B) Anthrax
(C) Canine distemper
(D) Trypanosomiasis

2016

154. Which of the following is an expected histopathological lesion in a rat suffering from severe chronic progressive nephropathy?
(A) Interstitial fibrosis, tubular dilation with protein casts, and glomerular sclerosis.
(B) Interstitial fibrosis, tubular dilation with protein casts, and glomerular sclerosis.
(C) Massive infiltration of the renal parenchyma by highly neoplastic lymphoid cells.
(D) Severe, acute suppurative pyelonephritis with ascending bacterial inflammation.

2025

155. Identify the correct pair associating the infectious agent with the resulting pathology in rabbits.
(A) Rabbit hemorrhagic disease virus - Hepatic coagulation necrosis
(B) Myxoma virus - Chronic caseous bronchopneumonia in the lungs
(C) Eimeria stiedae - Severe suppurative bacterial encephalitis
(D) Pasteurella multocida - Formation of cutaneous myxofibromas

Downloaded from www.pdf.tube

(B) The primary lesion is widespread cortical necrosis and severe malacia.
(C) The virus travels via retrograde transport along peripheral nerves.
(D) Negri bodies are classic eosinophilic intracytoplasmic inclusions.

2024
203. What is the fundamental difference between specific pathogen-free (SPF) and gnotobiotic laboratory animals?
(A) SPF animals lack specified pathogens with undefined flora, while gnotobiotic animals have a completely defined flora.
(B) SPF animals are raised in standard outdoor enclosures, whereas gnotobiotic animals are raised in strict barrier isolators.
(C) SPF animals possess a hyperactive immune system, whereas gnotobiotic animals lack any functional immune system.
(D) There is absolutely no difference between the two groups; the clinical and scientific terms are completely synonymous.

2012
204. Which of the following best describes the classical gross pathological lesion of Tyzzer's disease in laboratory rodents?
(A) Severe, diffuse hepatic lipidosis giving the liver a yellowish, highly friable appearance
(B) Massive enlargement of the spleen with multiple large, dark red nodules
(C) Multiple, miliary, pale necrotic foci scattered throughout the hepatic parenchyma
(D) Severe, fibrinous peritonitis with multiple adhesions between the intestinal loops

2021
205. The presence of a severe, acute, hemorrhagic enteritis with complete destruction of the intestinal crypts in a captive tiger is most strongly indicative of which viral disease?
(A) Canine Distemper
(B) Rabies
(C) Feline Infectious Peritonitis
(D) Feline Panleukopenia

2018
206. Which of the following is the hallmark histopathological finding of the cutaneous (dry) form of Avian Pox in wild birds?
(A) Intranuclear basophilic inclusion bodies in splenic macrophages
(B) Large, eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies) in epithelial cells
(C) Severe lymphocytic depletion with syncytial cell formation in the bursa of Fabricius
(D) Diffuse granulomatous inflammation with multinucleated giant cells containing acid-fast bacilli

2006
207. What is the most relevant consequence of an inverted calcium to phosphorus ratio (high phosphorus, low calcium) in the diet of a captive reptile?
(A) Rapid spontaneous regression of visceral gout lesions across multiple internal organ serosa.
(B) Stimulating parathyroid hormone release, causing bone resorption and secondary hyperparathyroidism.
(C) Massive deposition of calcium in soft tissues, particularly the kidneys and systemic blood vessels.
(D) Rapid development of severe, acute hepatic necrosis and widespread intravascular coagulation.

NODIA

2013
208. Which pathological feature provides the most accurate basis for diagnosing chlamydiosis in a wild bird?
(A) Large, extracellular fungal granulomas in the respiratory lungs and air sacs.
(B) Numerous basophilic intranuclear inclusion bodies within the host hepatocytes.
(C) Extravasated urate crystal deposition across multiple internal visceral organs.
(D) Fibrinous airsacculitis, hepatosplenomegaly, and elementary bodies in macrophages.

2002
209. A toxicological investigation of a poisoned wild canid reveals severe, widespread hemorrhage into body cavities and excessive bleeding from minor wounds. Which toxicant is the established cause of this syndrome?
(A) Anticoagulant rodenticide
(B) Organophosphate insecticide
(C) Strychnine toxicant
(D) Heavy metal lead

2022
210. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R).
Assertion (A): Specific Pathogen Free (SPF) rodents exposed to normal environmental flora may suffer severe clinical disease and high mortality.

by uric acid deposits.

2012

164. Which of the following best describes the classical histopathological lesion of rabies virus infection in the brain of a wild carnivore?
(A) Widespread ischemic necrosis of the cerebral cortex with prominent liquefactive malacia.
(B) Severe, acute purulent leptomeningitis with massive infiltration of polymorphonuclear neutrophils.
(C) Multifocal granulomatous inflammation forming distinct caseating tuberculous nodules in the cerebellum.
(D) Non-suppurative encephalomyelitis with eosinophilic Negri bodies in the hippocampus or Purkinje cells.

2007

165. The presence of 'tiger-stripe' hemorrhages in the colon and severe lymphoid depletion in a wild felid is most strongly indicative of which viral disease?
(A) Feline Herpesvirus
(B) Feline Panleukopenia
(C) Feline Calicivirus
(D) Rabies

2003

166. How does the etiology of Newcastle disease differ from that of avian influenza?
(A) Newcastle disease is a bacterial infection, whereas avian influenza is strictly viral.
(B) Newcastle disease affects water birds, whereas avian influenza only affects raptor species.
(C) Newcastle is caused by a Paramyxovirus, while avian influenza is caused by an Orthomyxovirus.
(D) There is no difference; both diseases are caused by completely identical viral strains.

2014

167. What is the most relevant consequence of a high-protein diet in aging laboratory rats?
(A) It successfully prevents the development of benign mammary fibroadenomas.
(B) It causes the rapid, spontaneous regression of systemic amyloidosis lesions.
(C) It leads directly to the development of clinical scurvy and bone fractures.
(D) It accelerates progression and severity of chronic progressive nephropathy.

2011

168. Which pathological feature provides the most accurate basis for classifying a lesion as pregnancy toxemia in a guinea pig?
(A) Severe hepatic lipidosis and an empty stomach in a late-gestation female
(B) Marked enlargement of costochondral junctions and subperiosteal bleeding
(C) Severe, diffuse caseous bronchopneumonia and fibrinous pleuritis
(D) Multiple benign fibroadenomas distributed within the mammary tissue

2005

169. A toxicological investigation of a wild herbivore reveals bright green fluid in the stomach, severe gastroenteritis, and acute kidney injury. The animal was found near an agricultural area. Which toxicant is suspected?
(A) Anticoagulant rodenticide (e.g., Brodifacoum)
(B) Arsenic-based herbicide/pesticide
(C) Cholecalciferol-based rodenticide
(D) Organophosphate insecticide

2017

170. In the following question, a statement of Assertion (A) is followed by a statement of Reason (R).
Assertion (A): Diclofenac toxicity caused a catastrophic decline in vulture populations across the Indian subcontinent.
Reason (R): Vultures lack the specific enzymatic capacity to properly metabolize diclofenac, leading to its accumulation, severe acute nephrotoxicity, and fatal visceral gout.
(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

2004

171. Identify the correct pair associating the specific viral disease of laboratory mice with its target tissue and characteristic lesion.
(A) Sendai virus - Respiratory epithelium, causing pneumonia
(B) Mousepox - Salivary glands, causing severe tissue necrosis
(C) Murine hepatitis virus - Epidermis, causing vesicle formation
(D) Sialodacryoadenitis virus - Hepatocytes, causing necrosis

Click Here to Buy book on Flipkart

2008

156. How does the etiology of murine mycoplasmosis differ from that of corynebacteriosis in laboratory rodents?

(A) Mycoplasmosis is caused by a highly infectious enveloped virus, whereas corynebacteriosis is caused by a fungus.

(B) Mycoplasmosis is caused by a parasitic nematode, whereas corynebacteriosis is caused by a Gram-negative bacillus.

(C) Mycoplasmosis is caused by wall-less bacteria, whereas corynebacteriosis is caused by Gram-positive bacilli.

(D) There is no difference between the two; both conditions are caused by completely identical bacterial species.

2025

157. What is the primary toxicological mechanism by which organophosphates cause mortality in free-ranging wildlife?

(A) They bind irreversibly to hemoglobin, causing cellular hypoxia.

(B) They cause massive hepatic necrosis and acute liver failure.

(C) They induce severe renal tubular necrosis resulting in uremia.

(D) They inhibit acetylcholinesterase, leading to respiratory failure.

2021

158. Which finding is LEAST relevant to a diagnosis of tuberculosis in a captive elephant?

(A) Chronic weight loss and poor body condition

(B) Caseocalcareous granulomas in the lungs and thoracic lymph nodes

(C) Severe acute vesicular stomatitis

(D) Positive culture for Mycobacterium tuberculosis from trunk washes

2017

159. The development of inclusion body disease in captive reptiles is frequently associated with infestation by which external parasite, serving as a potential vector?

(A) Ixodes scapularis (deer tick)

(B) Ophionyssus natricis (snake mite)

(C) Psoroptes cuniculi (ear mite)

(D) Sarcoptes scabiei (mange mite)

2008

160. Select the correct sequence for safely performing a necropsy on a non-human primate to minimize occupational hazards.

(A) Perform necropsy on open table → Wear standard scrubs → Hose down table → Dispose of tissues in regular trash

(B) Wash carcass vigorously → Don standard PPE → Dissect the organs rapidly → Discard protective gloves safely

(C) Defrost carcass in open room → Perform necropsy barehanded to maintain tactile sensitivity → Wash hands with soap

(D) Don complete PPE → Use biosafety cabinet → Moisten carcass → Decontaminate surfaces and instruments

2003

161. Which of the following inferences can be drawn if a wild carnivore is found dead with severe, diffuse, granulomatous pneumonia and characteristic spherules containing endospores on histopathology?

(A) The animal was infected with the dimorphic fungus Coccidioides immitis.

(B) The animal died of a severe viral infection such as Canine Distemper virus.

(C) The animal suffered from an advanced, long-standing tapeworm infestation.

(D) The animal died of acute bacterial septicemia and multi-organ failure.

2001

162. Identify the INCORRECT statement regarding the pathology of foot-and-mouth disease (FMD) in wild ruminants.

(A) The lesions primarily consist of vesicles and erosions on the oral mucosa and coronary bands.

(B) Young animals may die acutely from severe myocarditis (tiger heart).

(C) The disease typically causes a high mortality rate in adult populations.

(D) It is caused by an Aphthovirus within the family Picornaviridae.

2023

163. What is the fundamental difference between visceral gout and articular gout in captive birds?

(A) Visceral gout involves urate deposition on the serosal surfaces of internal organs (pericardium, liver), while articular gout involves urate tophi in the joints.

(B) Visceral gout is triggered by systemic viral infection, while articular gout is purely due to nutritional lack.

(C) Visceral gout occurs strictly in mammalian species, whereas articular gout occurs exclusively in avian species.

(D) Visceral gout is characterized by calcium deposits, while articular gout is characterized

NODIA

(C) Mousepox remains entirely asymptomatic in all strains, whereas MHV inevitably causes 100% mortality in infected hosts.
(D) There are absolutely no clinical or pathological distinctions between the two; the gross lesions are completely identical.

2005

180. Match the disease with its characteristic gross or microscopic lesion in the specified animal.

List I: (Disease and Animal Host)
P. Scurvy (Guinea Pig)
Q. Johne's Disease (Wild Ruminant)
R. Tyzzer's Disease (Mouse)
S. Myxomatosis (Rabbit)

List II: (Characteristic Lesion)
1. Miliary hepatic necrosis
2. Enlarged costochondral junctions with hemorrhage
3. Gelatinous subcutaneous nodules
4. Corrugated, thickened intestinal mucosa

(A) P-2, Q-4, R-1, S-3
(B) P-4, Q-2, R-1, S-3
(C) P-2, Q-4, R-3, S-1
(D) P-1, Q-3, R-4, S-2

2023

181. Identify the correct statement regarding the pathology of dirofilariasis in wild canids.
(A) The adult nematodes typically reside strictly within the left ventricle and descending aorta.
(B) Chronic infection leads to right-sided heart failure, hypertension, and vena cava syndrome.
(C) The microfilariae cause massive, irreversible destruction of the hepatic tissue parenchyma.
(D) The primary and most significant lesion is a severe, purulent and acute meningoencephalitis.

2006

182. What is the primary cause of the severe, caseous bronchopneumonia often seen in laboratory rodents infected with Corynebacterium kutscheri?
(A) Direct viral destruction of alveolar macrophages, leading to secondary bacterial invasion.
(B) Direct inhalation of the bacteria, leading to a severe and progressive viral-like pneumonia.
(C) Hematogenous bacterial spread from the GI tract, causing embolic pneumonia and abscesses.
(D) Secondary infection by parasitic lungworms that directly damage the airway defense mechanisms.

2008

183. A wild feline presents with severe, chronic weight loss. Necropsy reveals thickened, corrugated intestinal mucosa and enlarged mesenteric lymph nodes. Acid-fast bacilli are identified in macrophages within the lamina propria. Which bacterium is the most likely cause?
(A) Mycobacterium bovis
(B) Salmonella enterica bacteria
(C) Clostridium perfringens
(D) Mycobacterium paratuberculosis

2001

184. Which pathology is LEAST relevant when diagnosing lead poisoning in a free-ranging bird?
(A) Massive hepatic necrosis and hemorrhage
(B) Acid-fast intranuclear inclusions in renal tubular epithelial cells
(C) Emaciation and severe muscle atrophy
(D) Impaction of the proventriculus and gizzard

2019

185. What represents an exception to the normal gross anatomy of a laboratory mouse when conducting a necropsy?
(A) The presence of a large, pale, aglandular forestomach
(B) The lack of a gall bladder
(C) The presence of extensive brown fat deposits between the scapulae
(D) The presence of large, lobulated seminal vesicles in males

2010

186. Based on the pathophysiology of anticoagulant rodenticide toxicity, which diagnostic test on a liver sample from a deceased wild canid would definitively confirm the toxicant?
(A) Routine measurement of systemic acetylcholinesterase inhibition
(B) HPLC or mass spectrometry for specific anticoagulant compounds
(C) Highly sensitive PCR assay for viral hemorrhagic fever agents
(D) Atomic absorption spectroscopy for toxic heavy metal deposits

NODIA

2017

187. Identify the INCORRECT statement regarding avian influenza (AI).
(A) Waterfowl are primary reservoirs and usually show severe signs of highly pathogenic strains.

2019

172. What is the cause of the high mortality observed in juvenile Asian elephants infected with EEHV?

(A) Viral destruction of intestinal villi, leading directly to profuse and fatal diarrhea.

(B) Complete destruction of the bone marrow cellularity, leading to profound aplastic anemia.

(C) Destructive viral replication in capillary endothelial cells, causing hemorrhage and edema.

(D) Severe viral replication within the central nervous system causing acute fatal encephalitis.

2019

173. Which of the following inferences is most accurate regarding a captive reptile presenting with soft, pliable mandibles (rubber jaw) and spontaneous fractures of the long bones?

(A) The reptile is suffering from chronic infectious stomatitis, commonly known as mouth rot.

(B) The reptile has severe chronic lead poisoning with extensive systemic tissue accumulation.

(C) The reptile is infected with an arenavirus causing systemic inclusion body disease.

(D) The reptile has nutritional secondary hyperparathyroidism due to poor diet or low UV exposure.

2022

174. Which condition represents the most severe complication of an untreated infection with Psoroptes cuniculi in a laboratory rabbit?

(A) Spread of the mite to multiple internal organs, causing complete systemic failure.

(B) Transformation of the hyperkeratotic skin lesions into squamous cell carcinoma.

(C) Secondary bacterial infection causing otitis media, torticollis, and meningoencephalitis.

(D) Rapid development of severe hepatic coccidiosis throughout the liver parenchyma.

2022

175. The presence of marked biliary hyperplasia, heavily thickened bile ducts, and numerous intracellular protozoal stages in the biliary epithelium of a rabbit is diagnostic for:

(A) Tyzzer's infection

(B) Hepatic coccidiosis

(C) Hemorrhagic disease

(D) Myxoma viral disease

2007

176. Which of the following eliminates pasteurellosis as a primary diagnosis in a laboratory rabbit presenting with sudden death?

(A) The presence of severe, acute fibrinous pleuropneumonia and pleural effusion.

(B) The presence of a chronic, thick purulent exudate within the tympanic bulla.

(C) The presence of multiple caseous abscesses throughout the subcutaneous tissues.

(D) The presence of massive hepatic necrosis and coagulation with no respiratory lesions.

2008

177. Which statement accurately describes the etiopathology of babesiosis in wild canids?

(A) It is a tick-borne protozoal disease destroying erythrocytes, leading to anemia and icterus.

(B) It is a contagious viral disease causing severe, acute upper respiratory signs and distress.

(C) It is an infectious bacterial disease causing chronic, proliferative granulomatous enteritis.

(D) It is a systemic fungal disease causing severe, necrotizing and fatal meningoencephalitis.

2004

178. In the pathology of specific pathogen-free animals, what is the consequence if the barrier system fails and rats are exposed to Sialodacryoadenitis virus (SDAV)?

(A) Rapid development of fatal, necrotizing pneumonia in the entire colony, causing near total mortality within days.

(B) Massive, highly aggressive outbreak of multiple spontaneous epithelial tumors in extremely young animals.

(C) Cervical swelling, porphyrin staining ("red tears"), and photophobia, resolving in a few weeks but disrupting research.

(D) Profound, chronic immunosuppression leading to fatal opportunistic bacterial infections across multiple organ systems.

2024

179. What is the fundamental distinction in the pathology of mousepox compared to murine hepatitis virus (MHV)?

(A) Mousepox causes cutaneous lesions, extremity necrosis, and splenic necrosis, while MHV targets GI or liver, forming syncytia.

(B) Mousepox is caused by a highly infectious coronavirus, whereas MHV is caused by an extremely virulent orthopoxvirus strain.

Downloaded from www.pdf.tube

intestinal crypt epithelium.
(D) Widespread, chronic demyelination of the central nervous system.

2023
195. Identify the correct pair associating the disease, deficiency, or infectious agent with the resulting pathology in guinea pigs.
(A) Vitamin C deficiency - Defective collagen synthesis and periarticular hemorrhages.
(B) Pregnancy toxemia - Interstitial pneumonia and extensive fibrinous pleuritis.
(C) Cytomegalovirus - Hepatic necrosis and disseminated intravascular coagulation.
(D) Salmonella enterica - Cutaneous myxofibromas accompanied by severe rhinitis.

2019
196. How does the etiology of rabbit hemorrhagic disease (RHD) differ from that of myxomatosis?
(A) RHD is a bacterial disease, while Myxomatosis is a severe systemic opportunistic fungal disease.
(B) RHD strictly affects the respiratory tract, while Myxomatosis affects the gastrointestinal tract.
(C) There is absolutely no difference; both clinical conditions are caused by identical viral strains.
(D) RHD is a calicivirus causing hepatic necrosis, while Myxomatosis is a poxvirus causing skin tumors.

2013
197. What is the primary toxicological mechanism by which lead poisoning causes pathology in wildlife?
(A) It binds to acetylcholinesterase, leading to profound overstimulation of the nervous system.
(B) It disrupts enzyme systems involved in heme synthesis, causing anemia, neurotoxicity, and nephrotoxicity.
(C) It causes massive, acute hepatic necrosis and widespread disseminated intravascular coagulation.
(D) It acts strictly as a vitamin K antagonist, leading to severe, uncontrollable internal hemorrhage.

2005
198. Which finding is LEAST relevant to a diagnosis of Peste des Petits Ruminants in a wild goat?
(A) Severe, erosive stomatitis and enteritis
(B) Progressive bronchointerstitial pneumonia
(C) Vesicular lesions on the coronary bands
(D) Epithelial syncytial cell formation

NODIA

2005
199. The development of severe, chronic progressive nephropathy in aging laboratory rats is frequently associated with which management factor?
(A) Housing in a specific pathogen-free facility
(B) Chronic exposure to low carbon dioxide levels
(C) Provision of complex environmental enrichment
(D) Feeding diets high in protein and calories

2022
200. Consider the following statements about wild animal pathology:
I. Anthrax causes an enlarged, friable spleen and unclotted blood in herbivores.
II. Canine distemper virus causes demyelinating encephalomyelitis.
III. EEHV causes fatal hemorrhagic disease in elephants.
IV. Lead poisoning causes basophilic stippling in erythrocytes.
V. Capture myopathy is caused by a nutritional deficiency of Vitamin D.
Which of the above statement(s) is/are INCORRECT?
(A) V
(B) I, II and V
(C) III and IV
(D) II, III and V

2021
201. Which of the following inferences can be drawn if a captive macaque presents with severe weight loss, chronic coughing, and caseous granulomas in the lungs?
(A) The animal is infected with Macacine herpesvirus 1.
(B) The animal is infected with Mycobacterium tuberculosis, posing a severe zoonotic risk.
(C) The animal has scurvy due to Vitamin C deficiency.
(D) The animal is suffering from Simian Immunodeficiency Virus without any secondary infections.

2002
202. Identify the INCORRECT statement regarding the pathology of rabies.
(A) The virus is typically transmitted via saliva through a bite wound.

4. Paramyxoviridae (Morbillivirus)

(A) P-2, Q-1, R-4, S-3
(B) P-1, Q-2, R-3, S-4
(C) P-4, Q-3, R-1, S-2
(D) P-2, Q-4, R-1, S-3

2008

141. Identify the correct statement regarding Chlamydiosis (Psittacosis) in wild and zoo birds.

(A) It is primarily a localized skin infection that occurs without any systemic or internal organ involvement.
(B) It causes the formation of large, extracellular fungal granulomas within the thoracic and abdominal air sacs.
(C) It is transmitted exclusively through the bite of infected mosquitoes and other blood-sucking arthropod vectors.
(D) Chlamydia psittaci is an obligate intracellular bacterium that poses a significant zoonotic risk to humans.

2006

142. What is the primary cause of the severe, fatal enteritis seen in wild felids infected with feline panleukopenia virus?

(A) Toxin production by opportunistic bacterial invaders such as virulent strains of Clostridium perfringens.
(B) Viral replication restricted to apical enterocytes, leading to severe and acute malabsorptive diarrhea.
(C) Viral destruction of rapidly dividing cells in the crypts of Lieberkühn, causing villous collapse.
(D) Massive granulomatous inflammatory cell infiltration throughout the mucosal layers of the lamina propria.

2016

143. A laboratory rabbit presents with a head tilt (torticollis) and purulent nasal discharge. Necropsy reveals an accumulation of caseous pus in the tympanic bulla. Which bacterium is the most likely cause?

(A) Clostridium piliforme
(B) Pasteurella multocida
(C) Salmonella Typhimurium
(D) Corynebacterium kutscheri

2016

144. Which pathology is LEAST relevant when diagnosing hypovitaminosis A in captive birds?

(A) Squamous metaplasia of the mucous glands
(B) Hyperkeratosis of the skin and feet
(C) Formation of white pustules in the oral cavity and esophagus
(D) Diffuse thinning of the long bones and multiple fractures

2017

145. What represents an exception to the normal gross anatomy of a laboratory rat when conducting a necropsy?

(A) The presence of distinct palatine tonsils
(B) The presence of a large, lobulated liver
(C) The absence of a gall bladder
(D) The presence of a large, visible thymus in a young adult

2005

146. Based on the pathophysiology of capture myopathy, which clinical intervention would be most appropriate immediately following the capture of a wild herbivore to prevent this condition?

(A) Systemic administration of high doses of synthetic corticosteroids to suppress the active immune system.
(B) Administering a potent loop diuretic agent to force excess fluid directly out of injured muscle tissue.
(C) Minimizing pursuit time, providing immediate physical cooling, and administering supportive fluid therapy.
(D) Inducing deep, continuous general anesthesia across the animal for a minimum duration of 24 hours.

2005

147. Identify the INCORRECT statement regarding avian influenza (AI) in wild birds.

(A) Highly pathogenic strains always originate directly from bats before infecting birds.
(B) Waterfowl serve as the natural reservoir for many AI virus subtypes.
(C) Low pathogenic strains typically cause mild or asymptomatic respiratory or enteric infections.
(D) Highly pathogenic AI can cause sudden death with severe petechial hemorrhages in wild birds.

2001

148. Which is the most appropriate technique for preserving tissues intended for legal or forensic histopathology in wildlife cases?

(A) Freezing the specimens immediately in a laboratory freezer at -20°C.
(B) Immersing the collected tissue specimens in

Click Here to Buy book on Amazon

Reason (R): SPF rodents lack any functional T or B lymphocytes, making them inherently immunodeficient and unable to mount an immune response against common commensal organisms.

(A) Both A and R are true and R is the correct explanation of A.
(B) Both A and R are true but R is not the correct explanation of A.
(C) A is true but R is false.
(D) A is false but R is true.

****

NODIA

37. (A) Severe bronchiectasis and cobblestone lungs
Mycoplasma pulmonis is the primary etiological agent of murine respiratory mycoplasmosis. While both species can be infected, rats develop a more chronic, progressive, and destructive disease. In rats, the pathogen causes severe, extensive bronchiectasis and squamous metaplasia of the bronchial epithelium, leading to a highly characteristic gross "cobblestone" appearance of the lung lobes.

38. (A) Canine Distemper Virus
Canine distemper virus is a morbillivirus known for its unique ability to produce both eosinophilic intranuclear and intracytoplasmic inclusion bodies within the same cell. Additionally, it induces fusion of infected epithelial or lymphoid cells, resulting in the formation of large multinucleated syncytia. These histopathological features in respiratory tissues of tigers are diagnostic for this infection.

39. (B) Simian Immunodeficiency Virus - $CD4^+$ T lymphocyte depletion and lymphoid atrophy
Simian Immunodeficiency Virus is a lentivirus that infects non-human primates. It targets $CD4^+$ helper T lymphocytes and macrophages, leading to progressive cellular depletion and marked atrophy of lymphoid tissues. This profound loss of immune function results in acquired immunodeficiency syndrome, rendering the animal highly vulnerable to opportunistic infections and wasting.

40. (A) II
Sialodacryoadenitis virus is a coronavirus causing necrosis of salivary and lacrimal glands in rats. Corynebacterium kutscheri is a bacterium that causes pseudotuberculosis. Trichophyton mentagrophytes is a dermatophyte fungus causing ringworm. However, Demodex musculi is not a fungal organism but rather a microscopic parasitic mite that causes democidic mange in mice.

41. (D) Bacillus anthracis
Anthrax, caused by Bacillus anthracis, is an acute, septicemic disease of wild and domestic herbivores. The bacterium produces lethal toxins that damage vascular endothelium, causing widespread hemorrhage and failure of the blood-clotting mechanism. Typical post-mortem findings include dark, unclotted blood oozing from natural orifices, incomplete rigor mortis, and a severely enlarged, dark, friable spleen.

42. (A) They are known to be free of explicitly defined pathogenic microorganisms.
Specific pathogen-free laboratory rodents are maintained under strict biosecurity to ensure they do not harbor a defined list of pathogens. Unlike germ-free animals, they are intentionally colonized with a complex, non-pathogenic commensal microflora. This ensures normal physiological development while preventing the confounding experimental effects of infectious pathogens.

43. (B) Granulomatous encephalitis
Feline Panleukopenia, caused by a parvovirus, targets rapidly dividing cells. Pathological features include severe intestinal crypt epithelial necrosis, segmental necrotizing enteritis, and marked lymphoid depletion within lymphoid organs. Granulomatous encephalitis, characterized by macophages and giant cells, is not a feature of this acute viral disease, which instead affects tissues with high mitotic activity.

44. (B) They are benign, encapsulated masses that can grow to a massive size.
Mammary fibroadenomas are the most common spontaneous neoplasms in aging female Sprague-Dawley rats. These tumors are benign, well-encapsulated, and non-metastatic, although they can reach massive dimensions, sometimes exceeding the animal's body weight. They do not invade underlying abdominal muscle tissues or regress spontaneously, and they pose minimal risk of distant metastasis.

45. (A) It inhibits Vitamin K epoxide reductase, depleting active clotting factors.
Anticoagulant rodenticides act by inhibiting the enzyme Vitamin K epoxide reductase. This block prevents the recycling of active Vitamin K, which is an essential cofactor for the post-translational gamma-carboxylation of clotting factors II, VII, IX, and X. Depletion of these active factors impairs the coagulation cascade, causing widespread, fatal hemorrhage.

46. (C) Eimeria stiedae
Eimeria stiedae is the unique, highly host-specific protozoan parasite responsible for hepatic coccidiosis in rabbits. The sporozoites migrate to the liver and infect the epithelial cells

Downloaded from www.pdf.tube

Sprague-Dawley and Fischer 344 strains. The severity and progression of this glomerular and tubulointerstitial disease are highly influenced by diet. Formulations high in protein exacerbate renal damage by increasing glomerular filtration rates, intraglomerular pressure, and the metabolic workload on remaining functional nephrons.

10. (C) (A) is true but (R) is false.
Young rabbits under four to eight weeks old are resistant to rabbit hemorrhagic disease virus. This age-dependent resistance is a physiological characteristic related to the lack of mature viral receptors on young hepatocytes, rather than maternal antibodies. Thus, while young rabbits are indeed spared from clinical disease, the protection is not mediated by passive maternal immunity.

11. (B) Pasteurella multocida
Snuffles is a highly contagious respiratory disease in rabbits primarily caused by Pasteurella multocida, a Gram-negative bacterium. The pathogen colonizes the nasal passages, leading to mucopurulent rhinitis, sinusitis, and otitis media. In severe or chronic cases, the infection descends into the lower respiratory tract, resulting in fibrinopurulent pleuropneumonia, pulmonary abscessation, and systemic septicemia.

12. (B) Proline and lysine hydroxylation in collagen synthesis
Guinea pigs lack the enzyme L-gulonolactone oxidase and cannot synthesize ascorbic acid endogenously. Vitamin C is an essential cofactor for the enzymes prolyl hydroxylase and lysyl hydroxylase. These enzymes catalyze the hydroxylation of proline and lysine residues, which is essential for the stabilization of the triple helix structure of collagen fibers.

13. (A) Caseous granulomas in the lungs and bronchial lymph nodes
Tuberculosis in non-human primates, caused by Mycobacterium tuberculosis, is a severe, progressive disease. Transmission occurs primarily via the aerosol route, making the respiratory tract the primary site of infection. Pathological findings typically feature multifocal caseous granulomas with central necrosis, multinucleated giant cells, and acid-fast bacilli in the lung parenchyma and regional tracheobronchial lymph nodes.

NODIA

14. (C) Intracytoplasmic and intranuclear inclusions in respiratory epithelium
Canine distemper virus is a morbillivirus with pantropic properties, infecting epithelial, lymphoid, and nervous tissues. In wild carnivores like tigers, the presence of both eosinophilic intracytoplasmic and intranuclear inclusion bodies within epithelial cells of the respiratory, urinary, or gastrointestinal tract, as well as in astrocytes, is highly pathognomonic and serves as a definitive histopathological hallmark.

15. (D) Trypanosoma evansi - Severe purulent encephalitis
Trypanosoma evansi is a protozoan parasite causing Surra, which clinically presents with fever, anemia, edema, and nervous signs in wild carnivores. The neurological lesions typically consist of nonsuppurative meningoencephalitis characterized by perivascular cuffing of mononuclear cells, rather than purulent inflammation. Purulent encephalitis is associated with bacterial infections, not intravascular protozoan parasites.

16. (C) Peste des Petits Ruminants virus
Peste des Petits Ruminants is a highly contagious morbillivirus infection of small ruminants. It targets the digestive and respiratory tracts, causing mucosal necrosis and interstitial pneumonia. The classic clinical-pathological presentation includes erosive-ulcerative stomatitis, severe hemorrhagic enteritis leading to profuse diarrhea, and bronchointerstitial pneumonia, resulting in high morbidity and mortality in young wild ungulates.

17. (B) Capillary endothelial cells
Elephant Endotheliotropic Herpesvirus is a highly lethal pathogen affecting young Asian and African elephants. The virus exhibits a strict tropism for microvascular endothelial cells, particularly within cardiac, visceral, and mucosal capillaries. Replication within these cells causes widespread endothelial destruction, leading to generalized vascular leakage, extensive microvascular hemorrhages, severe edema, and rapid cardiac failure.

18. (D) Highly Pathogenic Avian Influenza
Highly Pathogenic Avian Influenza is a systemic viral disease of wild and domestic birds. The virus causes acute multi-organ failure through direct replication in vascular endothelial cells

and parenchymal cells. Typical gross pathological findings include widespread visceral congestion, petechial hemorrhages on epicardial surfaces, severe pulmonary edema, and multifocal necrosis within the pancreas, spleen, and liver.

19. (A) Uric acid
Birds are uricotelic organisms that excrete nitrogenous waste primarily as uric acid, synthesized in the liver. Since uric acid has low solubility in water, any impairment in renal function or severe dehydration hinders its clearance. This leads to hyperuricemia and the subsequent precipitation of insoluble white urate crystals, or tophi, on visceral surfaces.

20. (B) P-3, Q-2, R-1, S-4
Infectious stomatitis presents with oral mucosal petechiae and caseous exudate. Inclusion body disease in boid snakes features pathognomonic eosinophilic intracytoplasmic inclusions within neurons and other cells. Nutritional secondary hyperparathyroidism leads to fibrous osteodystrophy due to bone resorption. Gout is characterized by the physical deposition of white urate tophi within visceral organs and articular spaces.

21. (A) Both conditions feature massive skeletal muscle necrosis due to severe metabolic acidosis and ATP depletion.
Capture myopathy and exertional rhabdomyolysis share a common pathophysiological pathway driven by extreme overexertion. Anaerobic glycolysis in overstimulated muscles leads to intracellular lactic acid accumulation, severe local hyperthermia, and ATP depletion. This disrupts cell membrane integrity, causing widespread coagulative necrosis of skeletal myofibers, myoglobin release into circulation, and potential acute renal failure.

22. (D) Lead
Lead poisoning in waterfowl is a significant environmental hazard, typically occurring through the ingestion of spent ammunition or fishing sinkers. Absorbed lead disrupts heme synthesis, causing microcytic anemia with red blood cell basophilic stippling. Chronically, it damages renal proximal tubules, producing characteristic acid-fast intranuclear inclusion bodies representing complexes of lead and transport proteins.

23. (B) Pteropid fruit bats
Nipah virus is a zoonotic Henipavirus that causes severe encephalitis and respiratory illness in humans and domestic animals. Pteropid fruit bats, commonly known as flying foxes, serve as the natural evolutionary reservoir. The virus is maintained subclinically within bat populations and is shed in their saliva, urine, and partially consumed fruits, facilitating spillover.

24. (C) Proliferation of mesenchymal cells in a mucinous matrix
Myxomatosis, caused by Myxoma virus, is characterized by the development of gelatinous mucinous skin tumors, particularly around the head and anogenital region. Histologically, these lesions consist of a proliferative network of large, stellate mesenchymal cells, known as myxoma cells, embedded within an abundant, loose mucinous extracellular matrix rich in glycosaminoglycans.

25. (D) Viral replication in myocytes → Centripetal axonal transport → CNS replication → Centrifugal axonal transport to salivary glands
Following a bite, rabies virus undergoes initial localized replication within skeletal myocytes. The virus then accesses motor endplates to initiate centripetal retrograde axonal transport via peripheral nerves to the central nervous system. Following rapid replication in neurons, the virus undergoes centrifugal anterograde transport along cranial and autonomic nerves to the salivary gland epithelium.

26. (A) Severe acute diarrhea is consistently observed in all infected strains.
Salmonellosis in laboratory mice, primarily caused by Salmonella enterica, varies widely in presentation. Resistant mouse strains often develop subclinical, carrier states without overt symptoms. Even in susceptible strains, the clinical picture is characterized more by systemic septicemia, lethargy, splenomegaly, and focal hepatic necrosis rather than consistent, severe acute diarrhea across all strains.

27. (B) AA amyloid
Spontaneous amyloidosis in aging laboratory mice is typically secondary or reactive amyloidosis, classified as AA amyloid. This condition is characterized by the extracellular deposition of amyloid A protein fibrils, which are derived from serum amyloid A, an acute-phase

Click Here to Buy book on Flipkart

reactant synthesized in the liver in response to chronic inflammatory stimuli or age-associated tissue degradation.

28. (B) Whole blood and brain tissue
Organophosphate compounds exert toxicity by binding to and inactivating the acetylcholinesterase enzyme. For legal and forensic confirmation, specimens must be submitted to measure this enzyme inhibition. Brain tissue is the gold standard post-mortem sample as it reflects central nervous system inhibition, while whole blood is the preferred sample for assessing circulating cholinesterase levels.

29. (A) It is a tick-borne viral hemorrhagic fever affecting both monkeys and humans.
Kyasanur Forest Disease is caused by a flavivirus endemic to Karnataka, India. It is transmitted primarily by the bite of infected Haemaphysalis spinigera ticks. The disease causes severe, often fatal, hemorrhagic fever and encephalopathy in wild primates, which act as sentinel hosts, and can cause a similar, highly dangerous febrile illness in humans.

30. (A) I and II
Pregnancy toxemia in guinea pigs is a metabolic disorder characterized by severe hepatic lipidosis. Additionally, Vitamin C deficiency leads to impaired osteoid matrix synthesis, causing enlarged costochondral junctions. However, cytomegalovirus is typically subclinical in immunocompetent adult guinea pigs, only causing localized sialadenitis rather than strictly fatal systemic disease in all adult animals.

31. (D) Gross lesions are identical to Rinderpest, requiring molecular differentiation.
Peste des Petits Ruminants virus belongs to the Morbillivirus genus and shares high genetic and antigenic similarity with Rinderpest virus. In wild ruminants, both pathogens induce identical mucosal erosions, enteritis, and respiratory lesions. Consequently, gross pathology alone cannot differentiate between these two devastating viral diseases, necessitating confirmatory molecular or serological diagnostic assays.

32. (C) Interstitial pneumonia and bronchiolar necrosis
Sendai virus is a highly contagious parainfluenza virus that targets the respiratory tract of laboratory rodents. The primary lesions are concentrated in the lungs and airways, characterized by necrosis of the bronchiolar epithelium and a lymphohistiocytic interstitial pneumonia. This viral damage destroys the mucociliary apparatus, predisposing the animals to secondary bacterial infections.

33. (B) Renal tubular excretion of uric acid
Diclofenac is a non-steroidal anti-inflammatory drug that is highly toxic to vultures. It inhibits cyclooxygenase enzymes, which reduces renal blood flow and directly impairs the active secretory mechanisms of the renal tubular epithelial cells. This inhibition of renal excretion leads to acute hyperuricemia, systemic visceral gout, and extensive urate crystal deposition.

34. (D) A reptilian Arenavirus
Inclusion Body Disease is a significant transmissible disease affecting captive boid snakes, such as boas and pythons. The disease is caused by reptilian arenaviruses, which induce the formation of characteristic eosinophilic intracytoplasmic inclusion bodies within neurons, epithelial cells, and lymphoid cells. Affected snakes exhibit severe neurological dysfunction, chronic regurgitation, and wasting.

35. (B) II → I → IV → III
The physiological cascade of capture myopathy begins with extreme physical exertion and sympathetic exhaustion. This intense activity leads to localized muscle ischemia and a rapid shift to anaerobic glycolysis. The resulting lactic acidosis damages cell membranes, causing skeletal muscle necrosis. Finally, myoglobin released from damaged fibers is excreted, culminating in myoglobinuria and acute renal failure.

36. (C) The disease primarily causes severe caseous pneumonia in the lungs.
Johne's disease is a chronic granulomatous enteritis caused by Mycobacterium avium subspecies paratuberculosis. It is characterized by progressive weight loss, diarrhea, and thickened, corrugated intestinal mucosa with acid-fast bacterial clumps in macrophages. It does not primarily target the respiratory system or manifest as caseous pneumonia, which is instead a feature of tuberculosis.

Downloaded from www.pdf.tube

anthracis, characterized by incomplete rigor mortis, dark unclotted blood, and a liquefied spleen.

56. (C) An enlarged cecum due to accumulation of undegraded mucins
Germ-free rats lack a gastrointestinal microbiota, which normally degrades dietary fibers and endogenous mucins. Consequently, these undegraded macromolecules accumulate in the cecum, drawing water into the lumen via osmosis. This leads to marked cecal enlargement, which is a classic, highly predictable anatomical hallmark of axenic rodents compared to conventionally housed animals.

57. (D) Elephant Endotheliotropic Herpesvirus
Elephant Endotheliotropic Herpesvirus is a highly lethal double-stranded DNA virus that causes acute hemorrhagic disease primarily in young Asian elephants. The virus replicates within vascular endothelial cells, causing systemic microvascular damage. This leads to widespread internal hemorrhages, severe myocardial edema, pericardial effusion, and rapid cardiovascular collapse, resulting in extremely high mortality rates.

58. (A) Squamous metaplasia of the respiratory and salivary epithelium
Vitamin A is critical for maintaining the health and differentiation of epithelial tissues. In captive psittacines, hypovitaminosis A leads to the loss of normal secretory and ciliated cells, which are replaced by stratified squamous keratinizing epithelium. This squamous metaplasia typically affects the nasal passages, sinuses, salivary glands, and upper gastrointestinal tract.

59. (B) Syphacia obvelata
High mortality with concurrent diarrhea in a rat colony points to severe enteric pathogens. Murine Hepatitis Virus, Salmonella enterica, and Clostridium piliforme are well-documented causes of acute necrotizing enteritis and systemic mortality. In contrast, Syphacia obvelata is a pinworm parasite that typically causes subclinical infections with mild perianal irritation, but not severe diarrhea or mortality.

60. (A) P-4, Q-1, R-3, S-2
Corynebacterium kutscheri causes murine pseudotuberculosis, characterized by suppurative lesions in organs. Sialodacryoadenitis virus is a coronavirus that induces sialadenitis, presenting as cervical swelling and porphyrin staining. Aspiculuris tetraptera is a common pinworm parasite. Trichophyton mentagrophytes is a dermatophyte fungus responsible for circular, crusty ringworm lesions on the skin of rodents.

61. (A) Infection results in classic caseating granulomas in the lungs.
Tuberculosis is a highly significant disease in wild and captive non-human primates. Transmission typically occurs via aerosols, leading to pulmonary infection. The hallmark pathological finding is the development of classic caseating granulomas in the lung parenchyma and regional tracheobronchial lymph nodes, which contain central necrosis, macrophages, epithelioid cells, and acid-fast bacilli.

62. (C) Respiratory entry, local lymphoid replication, and systemic dissemination
Canine distemper virus pathogenesis begins with aerosol inhalation. The virus targets alveolar macrophages and bronchial lymph nodes to initiate primary replication. From these local lymphoid sites, infected mononuclear cells enter the blood, causing a primary viremia that distributes the virus to systemic lymphoid organs, followed by secondary replication in epithelial and nervous tissues.

63. (B) Chronic active hepatitis and hepatic portal fibroplasia with biliary hyperplasia
Helicobacter hepaticus is a spiral bacterium that persistently colonizes the cecum and colon of rodents. In susceptible strains of mice, such as A/J, the bacteria translocate to the liver via the portal system, causing chronic active hepatitis. This leads to progressive hepatic portal fibroplasia, lobular disorganization, and prominent biliary hyperplasia.

NODIA

64. (C) Babesiosis
Babesiosis is a tick-borne protozoan disease caused by Babesia species. The intraerythrocytic parasites multiply within host red blood cells, leading to direct mechanical damage and immune-mediated intravascular hemolysis. This process results in the classic clinical-pathological triad of severe regenerative hemolytic anemia, hemoglobinuria from hemoglobin clearance, and icterus from hyperbilirubinemia.

lining the bile ducts. This parasitic replication stimulates severe, marked papillomatous biliary hyperplasia, chronic portal inflammation, periductal fibrosis, and focal hepatic necrosis, impairing liver function.

47. (D) Fibrinous airsacculitis, hepatomegaly, and splenomegaly
Avian chlamydiosis, caused by Chlamydia psittaci, is a systemic bacterial infection affecting psittacines and other wild birds. The pathogen disseminates widely, producing classic gross lesions. These characteristically include diffuse fibrinous airsacculitis, fibrinous pericarditis, marked hepatomegaly with focal hepatic necrosis, and significant splenomegaly with a dark, congested, and friable parenchyma.

48. (C) RHD causes hepatic necrosis and DIC, whereas Myxomatosis causes cutaneous mucinous tumors.
Rabbit Hemorrhagic Disease is caused by a calicivirus and characterized by acute, diffuse hepatic necrosis and widespread disposable intravascular coagulation, leading to severe multi-organ hemorrhage. In contrast, Myxomatosis is a poxvirus infection that produces characteristic gelatinous, mucinous cutaneous swellings around the head, ears, and genitalia, accompanied by blepharoconjunctivitis.

49. (B) Deficiency of calcium or Vitamin D3, or an inverted Ca:P ratio, stimulates PTH release and bone resorption.
Nutritional Secondary Hyperparathyroidism in captive reptiles is driven by poor dietary calcium intake, lack of ultraviolet-B radiation needed for active Vitamin D3 synthesis, or an inverted $Ca{:}P$ ratio. The resulting chronic hypocalcemia continuously stimulates the parathyroid glands to secrete parathyroid hormone, inducing systemic osteoclastic bone resorption and replacement with fibrous tissue.

50. (C) (A) is true but (R) is false.
Maintaining a strict chain of custody is essential for any forensic or legal investigation involving poached wildlife. It ensures the integrity, identity, and security of the physical evidence from collection to the courtroom. However, the chain of custody refers to legal documentation and tracing, not the chemical preservation of the biological tissue samples.

51. (A) Crusty exudative otitis
Psoroptes cuniculi is the non-burrowing ear mite of rabbits. The mites inhabit the external ear canal, where their feeding and fecal antigens cause a hypersensitivity reaction. This leads to intense pruritus, inflammation, and the accumulation of a thick, brown, crusty exudate within the ear canal, potentially progressing to secondary bacterial otitis.

52. (C) The proventriculus and lymphoid tissue
Newcastle Disease Virus in its velogenic viscerotropic form is characterized by high mortality and prominent visceral lesions. The virus displays strong tropism for lymphoid and epithelial structures. Widespread hemorrhages and necrotizing lesions are characteristically located in the gastrointestinal tract, especially on the mucosal surfaces of the proventriculus, cecal tonsils, and intestinal lymphoid patches.

53. (A) The mouse is infected with a neurotropic strain of Murine Hepatitis Virus.
Certain neurotropic strains of Murine Hepatitis Virus, a coronavirus, exhibit a marked tropism for glial cells within the central nervous system. Infection leads to oligodendrocyte destruction, resulting in progressive demyelination of white matter tracts in the brain and spinal cord. This lesion serves as an experimental model for human demyelinating disorders.

54. (D) Tuberculosis is rarely a concern during necropsy of non-human primates due to natural immunity.
Tuberculosis is highly infectious and poses a major zoonotic threat during necropsies of non-human primates. Captive monkeys are highly susceptible to both Mycobacterium tuberculosis and Mycobacterium bovis. Aerosols generated during necropsy can easily transmit the bacteria to handlers, necessitating stringent biosafety measures, personal protective equipment, and regular health screenings for veterinary staff.

55. (B) Hemorrhagic Septicemia is caused by Pasteurella multocida, whereas Anthrax is caused by Bacillus anthracis.
Hemorrhagic Septicemia is a severe septicemic disease of wild and domestic ruminants caused by specific serotypes of Pasteurella multocida, manifesting as edematous swelling and acute fibrinous pleuropneumonia. In contrast, Anthrax is caused by the spore-forming bacterium Bacillus

Click Here to Buy book on Amazon

ANSWERS

1. (B) An animal that harbors a known, defined microflora and fauna, and is free of all other demonstrable microorganisms.
Gnotobiotic animals are defined as those with a completely known, defined microbiological status. They are either entirely germ-free or colonized with one or more specific, identified microorganisms. This precise control over the internal microbial environment is crucial for high-precision scientific research, minimizing confounding variables associated with variable commensal or pathogenic microflora populations in experimental cohorts.

2. (D) Synovial fluid
When investigating suspected systemic poisoning in wildlife, toxicological analysis prioritizes tissues where toxins accumulate or are processed. Stomach contents reveal recent ingestion, while the liver acts as the primary site of metabolic detoxification and storage. Adipose tissue accumulates lipophilic compounds. In contrast, joint fluid contains minimal metabolic activity and tissue mass, making it highly irrelevant for primary toxicology.

3. (A) Rapid osteoblastic activity
Alkaline phosphatase is a membrane-bound enzyme with multiple tissue-specific isoforms. In young, growing animals, including Wistar rats, bone development is extremely active. Osteoblasts produce high quantities of bone-specific alkaline phosphatase to facilitate mineralization of the skeletal matrix. Consequently, elevated serum levels represent a normal physiological state associated with skeletal growth rather than pathological organ damage.

4. (C) Q fever in personnel is most commonly associated with exposure to infected pregnant lagomorphs.
Q fever, caused by Coxiella burnetii, is a highly infectious zoonotic disease primarily associated with domestic ruminants such as sheep, goats, and cattle. The bacteria are shed in high numbers in birth products like placenta and amniotic fluid. Transmission from pregnant rabbits or other lagomorphs to facility personnel is extremely rare compared to ruminant exposures.

5. (A) Regional lymph nodes following skin inoculation
Ectromelia virus, the causative agent of mousepox, enters the host through abrasions in the skin. The virus initiates primary replication within the local dermis and rapidly spreads to regional lymph nodes. This initial lymphoid replication is a critical step in the pathogenesis, preceding the primary viremia that distributes the virus to the spleen and liver.

6. (D) They exhibit normal gastrointestinal tract morphology and physiological absorption rates.
Germ-free animals exhibit severe anatomical and physiological abnormalities, including an enlarged cecum, thin intestinal walls, and altered mucosal absorption due to the absence of microflora. Specific pathogen-free animals possess a normal, complex commensal microflora. This ensures normal development of the digestive tract and standard metabolic processing, making them superior models for translatable nutritional research.

7. (B) Multifocal miliary hepatic necrosis
Tyzzer's disease, caused by the intracellular bacterium Clostridium piliforme, affects a wide range of laboratory rodents. The pathogen typically enters via the gastrointestinal tract and disseminates to the liver through the portal circulation. This leads to a classic, highly characteristic triad of lesions including necrotizing hepatitis, focal necrotizing enteritis, and necrotizing myocarditis in susceptible hosts.

NODIA

8. (C) Cellophane tape test applied to the perianal region
Syphacia obvelata is a common pinworm infecting laboratory mice. Unlike other parasites, the adult females migrate out of the anus to deposit their eggs on the perianal skin. Utilizing a cellophane tape test allows for the direct physical collection of these adhesive eggs from the skin surface, serving as the most reliable diagnostic detection method.

9. (D) High protein intake
Chronic progressive nephropathy is a common age-related disease in rats, particularly

65. (A) Trichosomoides crassicauda
Trichosomoides crassicauda is a unique nematode parasite that resides in the transitional epithelium of the urinary bladder and renal pelvis of laboratory rats. The presence of the parasite, particularly the adult females, stimulates chronic physical irritation, leading to epithelial hyperplasia and transitional cell papillomas, which can predispose the animal to tumor development.

66. (D) Petechial hemorrhages, edema, and meningoencephalitis
Trypanosoma evansi is a vascular protozoan parasite causing Surra, which affects a wide range of mammals. In wild carnivores like tigers, the disease presents as an acute or chronic septicemia. The parasite damages vascular endothelium, leading to extensive petechial hemorrhages, generalized subcutaneous and laryngeal edema, and nonsuppurative meningoencephalitis due to perivascular inflammatory infiltration.

67. (A) They typically cause high mortality in their natural bat reservoir hosts.
Coronaviruses are highly prevalent in wildlife, with bats serving as key evolutionary reservoirs. Due to a highly adapted host-parasite relationship, bats generally carry and shed these viruses subclinically without developing significant disease or high mortality. Pathological consequences, severe respiratory disease, and systemic organ failure typically only manifest following spillover into aberrant intermediate or final hosts.

68. (C) Vesicular lesions on the oral mucosa and interdigital clefts
Foot-and-mouth disease is a highly contagious aphthovirus infection of cloven-hoofed wild and domestic ungulates. The virus exhibits a strong epitheliotropic tropism. It induces ballooning degeneration of epithelial cells in the stratum spinosum, resulting in the formation of classic fluid-filled vesicles and erosions on the oral mucosa, dental pad, tongue, and interdigital clefts.

69. (A) Diclofenac
Diclofenac is a non-steroidal anti-inflammatory drug that enters the vulture food chain through livestock carcasses. Vultures lack functional mechanisms to tolerate the drug, which causes acute renal failure by reducing renal blood flow. This leads to the rapid accumulation of uric acid in the blood, culminating in severe, fatal visceral gout and widespread urate deposition.

70. (A) I and II
Murine hepatitis virus is a coronavirus belonging to the Coronaviridae family. Sendai virus damages the airway mucociliary escalator, predisposing rodents to secondary bacterial pneumonia. However, ectromelia virus, causing mousepox, is not universally fatal; its clinical presentation and mortality rates vary dramatically depending on the specific genetic strain of the infected host mice.

71. (B) Severe vesicular glossitis
Lead toxicity in waterfowl impairs the gastrointestinal and nervous systems, leading to proventriculus impaction, peripheral nerve demyelination, and weakness. Chronically, it produces diagnostic acid-fast intranuclear inclusions within renal tubular cells. However, it does not cause severe vesicular glossitis, which is instead a characteristic lesion of viral epitheliotropic infections like foot-and-mouth disease.

72. (C) Utilizing a biosafety cabinet and wearing full personal protective equipment
Macacine herpesvirus 1, carried latently by macaques, causes a highly fatal encephalomyelitis in humans. Because necropsies involve aerosol generation and exposure to bodily fluids, they pose an extreme biosafety hazard. To prevent lethal zoonotic transmission, procedures must be conducted within a biosafety cabinet under negative pressure, utilizing full personal protective equipment including respirators.

73. (D) Trichophyton mentagrophytes is a common etiology with zoonotic potential.
Dermatophytosis in laboratory rodents is predominantly caused by the filamentous fungus Trichophyton mentagrophytes. Infected animals often remain subclinical or develop dry, crusty, circular alopecic lesions, which are typically concentrated on the head, neck, and limbs. Because of its zoonotic nature, this fungal pathogen represents a significant occupational risk for animal handlers.

Downloaded from www.pdf.tube

74. (A) Extracellular eosinophilic material that stains with Congo Red showing green birefringence
Amyloidosis is characterized by the extracellular deposition of insoluble, proteinaceous fibrillar sheets in various organs. In histological sections, these deposits appear as amorphous, eosinophilic material. Under polarized light microscopy, amyloid exhibits a highly diagnostic apple-green birefringence when stained with Congo Red dye, differentiating it from other hyaline or proteinaceous accumulations.

75. (B) Myoglobinuria from exertional rhabdomyolysis
Capture myopathy in translocated wild herbivores involves intense physical exertion, causing extensive anaerobic glycolysis and metabolic acidosis. This induces acute coagulative necrosis of skeletal myofibrils. The rapid breakdown of muscle cells releases massive quantities of myoglobin into the systemic circulation, which is filtered by the kidneys, resulting in the excretion of dark brown urine.

76. (B) Velogenic viscerotropic
Newcastle disease virus strains are categorized into pathotypes based on their systemic virulence. The velogenic viscerotropic pathotype is highly lethal and primarily targets mucosal and lymphoid tissues of the gastrointestinal system. Pathological examination reveals prominent necrotizing and hemorrhagic lesions, particularly within the mucosal lining of the proventriculus, Peyer's patches, and cecal tonsils.

77. (A) Ectromelia virus
Ectromelia virus is an orthopoxvirus that causes mousepox in laboratory mice. The virus infects epidermal cells, inducing characteristic large, eosinophilic intracytoplasmic inclusion bodies, known as Guarnieri bodies. These inclusions contain viral replication machinery and are diagnostic during histopathological evaluation of the cutaneous lesions, which typically present as ulcerative dermatitis.

78. (C) Hemorrhagic fever causing gastrointestinal bleeding and shock
Kyasanur Forest Disease virus is a flavivirus that targets vascular endothelial cells and lymphoid organs in wild primates. The infection leads to a severe hemorrhagic fever syndrome characterized by widespread capillary leakage, extensive gastrointestinal hemorrhage, and thrombocytopenia. This severe vascular disruption and blood loss ultimately culminate in systemic cardiovascular shock and high mortality.

79. (D) Demodex musculi
Demodex musculi is a microscopic follicular mite of laboratory mice. While immunocompetent mice usually harbor the parasite subclinically, immunocompromised individuals cannot control the mite population. This leads to heavy infestations within the hair follicles, resulting in progressive alopecia, severe scaling, epidermal hyperkeratosis, and chronic dermatitis, particularly on the head and neck.

80. (B) III and IV
Suspected anthrax carcasses must never be opened, as oxygen exposure stimulates the vegetative bacilli to form highly resilient spores. Diagnosing anthrax in wild herbivores utilizes peripheral blood or ear vein smears to identify the capsule. For legal investigations, a strict, documented chain of custody is mandatory for all forensic diagnostic samples.

81. (B) The external ear canal
Psoroptes cuniculi is the non-burrowing ear mite of rabbits that causes otoacariasis. The mites reside, feed, and reproduce directly within the lumen of the external ear canal. Consequently, when diagnosing this parasitic infestation, the external ear canal and its associated crusty exudative debris represent the primary and most yield-effective anatomical sampling site.

NODIA

82. (D) It is caused by a Leporipoxvirus, transmitted by fleas and mosquitoes.
Myxomatosis is a severe disease of rabbits caused by Myxoma virus, a genus-specific Leporipoxvirus. The virus is primarily transmitted mechanically via biting arthropod vectors, such as fleas and mosquitoes. It induces cutaneous mesenchymal cell proliferation, resulting in gelatinous, mucinous swellings of the face and genitals, accompanied by severe blepharoconjunctivitis.

83. (A) Gout in reptiles - Excessive retention of urea
Gout in reptiles is a metabolic disorder characterized by hyperuricemia and the deposition of insoluble sodium urate crystals, not urea. Because reptiles are uricotelic, they excrete nitrogenous waste primarily as uric acid.

Impaired renal function or dehydration results in the systemic accumulation and precipitation of uric acid, leading to visceral or articular gout.

84. (C) Simian Immunodeficiency Virus
Simian Immunodeficiency Virus is a lentivirus that infects non-human primates. It displays a selective tropism for $CD4^+$ T helper lymphocytes and macrophages. Progressive replication of the virus leads to systemic lymphoid depletion and a loss of cellular immunity, mirroring the pathogenesis of human HIV and predisposing the monkeys to opportunistic secondary infections.

85. (A) Inhibiting acetylcholinesterase, causing overstimulation of cholinergic receptors
Organophosphate compounds are potent neurotoxins that bind to the active site of the enzyme acetylcholinesterase. This binding prevents the degradation of the neurotransmitter acetylcholine within the synaptic cleft. The resulting accumulation of acetylcholine leads to continuous, overstimulation of muscarinic and nicotinic receptors, causing autonomic dysfunction, muscle tremors, respiratory failure, and death.

86. (D) Thickened glomerular basement membranes and protein casts
Chronic progressive nephropathy is a common age-related renal disease in laboratory rats. Histopathological hallmarks of this degenerative disease include marked thickening of the glomerular basement membranes, mesangial expansion, tubular atrophy, interstitial fibrosis, and the presence of abundant, large, eosinophilic proteinaceous casts within the lumina of dilated renal tubules.

87. (B) Risk of exposure to Mycobacterium tuberculosis, requiring immediate staff screening
Mycobacterium tuberculosis is a highly infectious zoonotic pathogen that poses a major occupational health hazard. Captive chimpanzees are highly susceptible, and post-mortem manipulation of an infected carcass can generate infectious aerosols. Thus, finding caseous pulmonary granulomas with acid-fast bacilli requires immediate biosafety protocols and comprehensive health screening of all exposed veterinary personnel.

88. (C) Acute hepatic necrosis leading to DIC and hemorrhage
Rabbit Hemorrhagic Disease Virus is a highly virulent calicivirus with a strict tropism for hepatocytes. The virus induces acute, diffuse lytic necrosis of the liver parenchyma, which impairs hepatic synthesis of clotting factors. This massive necrosis triggers a systemic cascade of disseminated intravascular coagulation, causing widespread hemorrhages and rapid, fatal circulatory shock.

89. (D) Cyanosis of the comb and wattles, with petechiae on visceral organs
Highly Pathogenic Avian Influenza is a systemic viral disease that infects vascular endothelial cells. This endothelial damage causes generalized vascular congestion, leakage, and thrombosis. Typical gross pathological findings in birds include cyanosis of the comb, wattles, and facial skin, subcutaneous edema of the head, and widespread petechial hemorrhages on visceral organs.

90. (A) Both (A) and (R) are true and (R) is the correct explanation of (A).
Specific pathogen-free animals are explicitly defined by the absence of a designated list of pathogenic microorganisms. However, they are not germ-free. They are raised with a normal, complex commensal microflora that is essential for standard gastrointestinal development, whereas germ-free animals are completely axenic, lacking any detectable microflora.

91. (B) It results in a severe, highly fatal gastroenteritis in adult rats.
Sialodacryoadenitis virus is a highly contagious coronavirus of laboratory rats. The virus targets the ductal epithelium of salivary and lacrimal glands, causing acute necrotizing sialadenitis and dacryoadenitis, which manifests as cervical swelling and ocular porphyrin staining. It does not cause fatal gastroenteritis in adult rats, which is typically associated with enteric rotaviruses.

92. (B) Aspiculuris tetraptera
Aspiculuris tetraptera is a common pinworm infecting the large intestine of laboratory mice. Unlike Syphacia species, the adult female worms do not migrate to the perianal skin to deposit eggs, but instead lay them directly within the colon. Consequently, diagnosis relies on fecal

Click Here to Buy book on Flipkart

flotation or colonic examination to detect the characteristic symmetrical ova.

93. (A) Do not open the carcass; take a peripheral blood smear and inform authorities.
Opening a carcass suspected of anthrax is strictly prohibited, as exposure to atmospheric oxygen triggers Bacillus anthracis to form highly resilient, infective spores that contaminate the environment. Diagnosis must be achieved by examining a peripheral blood smear, which contains abundant vegetative bacilli, and the appropriate regulatory authorities must be immediately notified.

94. (C) Renal tubular necrosis and osteomalacia
Cadmium bioaccumulation in wildlife causes multi-organ toxicity, primarily targeting the kidneys and skeleton. The metal accumulates in the renal cortex, damaging proximal tubular epithelial cells and inducing chronic necrosis. This renal impairment disrupts calcium and phosphorus metabolism, leading to secondary osteomalacia, characterized by bone demineralization and skeletal fragility.

95. (D) Negative energy balance leading to fat mobilization and ketogenesis
Pregnancy toxemia in guinea pigs is a metabolic emergency during late gestation, often triggered by stress or fasting. A sudden, severe negative energy balance forces the rapid mobilization of free fatty acids from adipose tissue. This overwhelming influx of lipids to the liver results in profound hepatic lipidosis, systemic ketosis, acidosis, and death.

96. (C) The macaque is a latent carrier and poses a zoonotic risk to human handlers when stressed.
Macacine herpesvirus 1 typically causes subclinical or mild, self-limiting oral mucosal lesions in its natural macaque hosts. However, the virus establishes lifelong latency in sensory ganglia. When stressed, infected macaques can shed the virus in saliva and ocular secretions, posing a highly lethal zoonotic threat of encephalomyelitis to exposed human handlers.

97. (B) Treating infected animals to eliminate the carrier state.
Mycoplasma pulmonis causes chronic, persistent murine respiratory mycoplasmosis. While antibiotics can temporarily alleviate clinical signs, they are completely ineffective at eliminating the persistent chronic carrier state in laboratory rodents. Consequently, treatment is not a viable strategy; control relies on maintaining SPF barriers, rigorous quarantine, diagnostic screening, and depopulation.

98. (D) The high susceptibility of germ-free animals to common environmental pathogens
Germ-free mice are reared in a completely sterile environment and possess an extremely underdeveloped mucosal immune system. They have smaller lymphoid organs, lower immunoglobulin levels, and lack a protective commensal microflora. Consequently, accidental exposure to conventional housing introduces common environmental pathogens, resulting in rapid, fatal infections due to their profound susceptibility.

99. (B) Systemic hemorrhages, multifocal splenic and hepatic necrosis, and cutaneous ulceration
Ranavirus is a highly lethal pathogen affecting amphibians and reptiles. The virus targets endothelial cells and parenchymal organs, inducing systemic necrotizing vasculitis. Typical pathological findings include widespread petechial and ecchymotic hemorrhages, multifocal areas of necrosis in the spleen, liver, and kidneys, along with severe, ulcerative skin lesions and systemic edema.

100. (B) P-2, Q-3, R-4, S-1
Feline Panleukopenia causes acute necrotizing enteritis with crypt necrosis. Elephant Endotheliotropic Herpesvirus replicates in endothelial cells, causing myocardial edema. Johne's Disease induces chronic granulomatous enteritis characterized by macrophages containing acid-fast bacilli. Peste des Petits Ruminants is characterized by an erosive-ulcerative stomatitis accompanied by severe, diffuse bronchointerstitial pneumonia.

NODIA

101. (A) An acute non-infectious muscle disease triggered by extreme stress of pursuit and capture.
Capture myopathy is a severe, non-infectious metabolic muscle disease of wild animals, particularly herbivores, triggered by the intense stress and physical overexertion of pursuit, restraint, or translocation. Extreme muscle activity leads to anaerobic glycolysis, massive lactic acidosis, hyperthermia, and widespread

coagulative necrosis of skeletal and cardiac myofibers.

102. (D) HS presents with edematous swelling of the head, neck, brisket, and petechial hemorrhages.
Hemorrhagic Septicemia is an acute, fatal septicemic disease of wild and domestic ruminants. The causative Pasteurella multocida strains release endotoxins that damage vascular endothelial cells. This induces widespread vascular leakage, manifesting as extensive, gelatinous edematous swelling of the submandibular region, neck, and brisket, accompanied by petechial and ecchymotic hemorrhages.

103. (B) Extensive hemocoelom and hematomas
Anticoagulant rodenticides disrupt the recycling of active Vitamin K, which is required for synthesizing functional clotting factors II, VII, IX, and X. In wild birds, this leads to a severe coagulopathy characterized by widespread vascular leakage and hemorrhage. Typical gross lesions include extensive blood accumulation within the coelomic cavity and subcutaneous hematomas.

104. (C) Suppurative bronchopneumonia, catarrhal enteritis, and enamel hypoplasia
Canine distemper virus is a pantropic morbillivirus that targets epithelial and lymphoid tissues. In wild canids, the infection causes immunosuppression, predisposing them to secondary bacterial infections. Typical gross pathological findings include severe suppurative bronchopneumonia, catarrhal enteritis, hyperkeratosis of the footpads, and characteristic enamel hypoplasia in young, developing animals.

105. (A) II → I → IV → III
Nutritional secondary hyperparathyroidism in reptiles begins with a dietary calcium deficiency or inverted calcium-to-phosphorus ratio. This chronic mineral imbalance stimulates the parathyroid glands to hypersecrete parathyroid hormone. Increased hormone levels induce osteoclastic bone resorption to mobilize skeletal calcium, leading to weakening and subsequent replacement of bone with fibrous tissue.

106. (B) Real-time PCR of oropharyngeal swabs or target tissues
Real-time polymerase chain reaction is the primary diagnostic method used to confirm active Nipah virus infection in wildlife. This molecular assay is highly sensitive and specific, allowing for rapid detection of viral RNA in target tissues or oropharyngeal swabs. It provides a reliable diagnosis crucial for managing spillover events and preventing potential zoonotic transmission.

107. (A) The virus travels to the central nervous system via hematogenous spread.
Rabies virus is highly neurotropic and travels to the central nervous system via retrograde axonal transport along peripheral nerves, rather than through the bloodstream. Hematogenous spread does not occur during rabies pathogenesis. The virus replicates locally in muscle tissue before entering sensory or motor nerves, eventually reaching the spinal cord and the brain.

NODIA

108. (C) The team must halt the necropsy and handle the carcass as a suspected Bacillus anthracis case.
Sudden death, incomplete rigor mortis, and blood exuding from natural orifices in a captive rhinoceros are classic signs of anthrax. Opening the carcass releases highly resilient spores of Bacillus anthracis into the environment, creating a severe biological hazard. To prevent widespread contamination and human exposure, the necropsy must be halted immediately and biosafety protocols enacted.

109. (D) Defective purine metabolism or renal impairment leading to hyperuricemia and joint deposition.
Articular gout in birds is primarily driven by hyperuricemia, which results from either defective purine metabolism or impaired renal excretion of uric acid. Birds lack the uricase enzyme, meaning uric acid is their primary nitrogenous waste product. When blood levels exceed solubility limits, microscopic monosodium urate crystals precipitate within the synovial membranes and joints, causing inflammation.

110. (B) III
Myxoma virus is a poxvirus that primarily causes cutaneous myxomatosis, characterized by gelatinous skin nodules, facial edema, and bleaconjunctivitis, rather than hemorrhagic enteritis. Meanwhile, Pasteurella multocida causes severe respiratory tract infections, otitis media, and torticollis, while rabbit hemorrhagic disease virus specifically infects hepatocytes, leading to acute hepatic necrosis and disseminated intravascular coagulation.

111. (B) The intermediate host allows replication and mutation, facilitating adaptation for human infection.
A coronavirus spillover from bats to an intermediate host provides an evolutionary environment where the virus replicates and undergoes genetic mutations. This intermediate step facilitates viral adaptation to new cellular receptors, such as angiotensin-converting enzyme two, making the virus highly capable of infecting humans. It acts as an essential bridge for zoonotic transmission and pandemic emergence.

112. (D) Newcastle Disease - Non-suppurative encephalomyelitis and lymphoid necrosis
Newcastle disease, caused by avian paramyxovirus serotype one, typically presents with nonsuppurative encephalomyelitis and widespread lymphoid necrosis as key histopathological features. Avian influenza does not produce eosinophilic intracytoplasmic inclusions. Psittacosis is a bacterial disease, and Marek's disease is characterized by peripheral nerve infiltration with neoplastic T lymphocytes, rather than fibrous osteodystrophy.

113. (A) Mouse hepatitis virus
Mouse hepatitis virus is a highly contagious coronavirus that causes a mild infection in immunocompetent mice, but leads to chronic, severe wasting disease and systemic failure in nude athymic mice. Because athymic mice lack functional T lymphocyte mediated immunity, they cannot clear the viral pathogen, resulting in persistent replication within the liver, intestines, and lymphoid tissues.

114. (C) Eosinophilic meningoencephalitis
Mycoplasma pulmonis is the primary causative agent of murine respiratory mycoplasmosis in laboratory rats, characterized by suppurative rhinitis, otitis media, tracheitis, and bronchiectasis with purulent exudate. Eosinophilic meningoencephalitis is not associated with this bacterial infection. Mycoplasma pulmonis primarily targets mucosal surfaces of the respiratory tract and middle ear, causing chronic, progressive inflammatory lesions.

115. (A) Cyanide
Cyanide poisoning in wild animals classically presents with bright red mucous membranes and cherry red venous blood due to the inhibition of cytochrome c oxidase, which prevents oxygen utilization by tissues. The distinct odor of bitter almonds from stomach contents is highly characteristic of cyanide ingestion. These postmortem findings differentiate cyanide toxicity from other chemical agents.

116. (D) Canids typically develop heavy worm burdens, while felines exhibit severe respiratory signs from very few worms.
In wild canids, Dirofilaria immitis infections typically present with high worm burdens in the pulmonary arteries and right heart, leading to chronic congestive heart failure. Conversely, domestic felines are highly susceptible to severe, acute respiratory disease, known as heartworm associated respiratory disease, which is triggered by the presence of only a very few adult worms.

117. (B) An arenavirus infection of boid snakes characterized by eosinophilic intracytoplasmic inclusions in CNS and other tissues.
Inclusion body disease in boid snakes is caused by reptarenavirus infections. The condition is characterized histopathologically by the presence of large, eosinophilic, intracytoplasmic inclusion bodies within neurons of the central nervous system, epithelial cells of the kidneys, hepatocytes, and lymphoid cells. It typically leads to progressive neurological dysfunction, secondary infections, and wasting.

118. (C) It causes acute, highly fatal hemorrhagic disease in juvenile Asian elephants.
Elephant endotheliotropic herpesvirus is a double stranded DNA virus that causes acute, highly fatal hemorrhagic disease primarily in juvenile Asian elephants. The virus targets vascular endothelial cells, leading to systemic capillary leakage, widespread visceral hemorrhage, and severe myocardial damage. This disease represents a major conservation challenge due to its rapid onset and high mortality rates.

119. (D) Acute renal failure leading to visceral gout
Diclofenac exposure in wild birds, particularly Gyps vultures, causes severe acute renal tubular necrosis. The inability of the kidneys to excrete uric acid leads to marked hyperuricemia. Consequently, white, chalky monosodium urate crystals deposit on the serosal surfaces of internal organs, including the pericardium, liver, and kidneys, culminating in fatal visceral gout.

Downloaded from www.pdf.tube

120. (A) I and II
Feline panleukopenia virus infects rapidly dividing tissues, leading to myeloid depletion and necrosis of intestinal crypts in wild felids. Canine distemper virus, a morbillivirus, also crosses species barriers to cause fatal neurological and respiratory disease in large felids. Trypanosoma evansi is a protozoan parasite, and rabies virus causes both furious and dumb forms of encephalomyelitis.

121. (B) Advancing age and genetic strain
In specific pathogen-free laboratory rodents, systemic amyloidosis is primarily a spontaneous degenerative disease linked to advancing age and genetic background. Amyloid deposits, consisting of misfolded proteins, accumulate in the extracellular space of organs like the kidneys, spleen, and liver. This condition is not related to dietary vitamins, heavy metal toxicity, or acute viral infections.

122. (B) Benign proliferation of glandular epithelium and fibrous stroma.
Mammary fibroadenoma is the most common benign mammary tumor in female laboratory rats, characterized by the simultaneous proliferation of both glandular epithelium and fibrous connective tissue stroma. These tumors can reach a massive size, yet they remain encapsulated and benign, without invading local lymph nodes, causing purulent inflammation, or developing caseous necrosis in the ducts.

123. (A) It causes acute, massive hepatic necrosis as its primary lesion.
Pasteurella multocida in rabbits primarily targets the respiratory system and middle ears, causing mucopurulent rhinitis, otitis media, and fibrinous pneumonia. It can spread hematogenously to form abscesses in organs like the liver, but it does not cause acute, massive hepatic necrosis as its primary lesion, which is characteristic of rabbit hemorrhagic disease virus instead.

124. (C) Their defined microflora allows controlled studies of host-pathogen interactions without interference.
Gnotobiotic animals are raised under sterile conditions so that all of their microflora are completely defined or absent. This status provides a clean baseline that allows researchers to study host pathogen interactions, immunology, and metabolic processes with high precision, removing the confounding variables introduced by undefined commensal bacteria present in standard laboratory animal facilities.

125. (D) Eimeria intestinalis
Infected rabbits with Eimeria intestinalis develop raised, white, multifocal lesions on the serosal surface of the intestines. These macroscopic lesions represent areas of severe coccidiosis with marked mucosal hyperplasia and congestion. This is distinct from hepatic coccidiosis, caused by Eimeria stiedae, which produces white, multifocal nodules restricted entirely to the biliary system of the liver.

126. (C) DDT and its metabolites
Organochlorine compounds like dichlorodiphenyltrichloroethane and its metabolites, such as DDE, interfere with calcium deposition during eggshell formation in birds. This inhibition of calcium adenosine triphosphatase in the shell gland results in thin shelled eggs that break easily under the weight of nesting parents, leading to severe reproductive failure and population declines in wild raptors.

127. (B) A large, thin-walled cecum occupying much of the abdomen
During the necropsy of a normal, healthy guinea pig, a highly prominent, thin walled cecum containing green, semi fluid digesta is always observed occupying a major portion of the abdominal cavity. This is a normal anatomical feature required for hindgut fermentation in herbivores and must not be misinterpreted as pathological expansion, distension, or tympany.

NODIA

128. (D) Tyzzer's causes multiple pale, miliary necrotic foci, while Salmonella causes hepatosplenomegaly with focal histiocytic nodules.
At necropsy, Tyzzer's disease, caused by Clostridium piliforme, presents with classic miliary, pale necrotic foci scattered throughout the liver tissue. In contrast, Salmonella enterica infection typically results in diffuse hepatosplenomegaly with small focal histiocytic aggregates or paratyphoid nodules. These distinct macroscopic patterns help differentiate the two bacterial infections before histopathological confirmation.

and lactic acidosis, protecting muscle tissue and preventing acute renal failure due to myoglobinuria.

147. (A) Highly pathogenic strains always originate directly from bats before infecting birds.
Highly pathogenic avian influenza strains do not originate from bats. Instead, these virulent strains emerge from low pathogenic precursors through mutation or reassortment within domestic poultry or wild bird populations. Waterfowl serve as the primary natural reservoir hosts for low pathogenic strains, while highly pathogenic avian influenza causes severe systemic lesions and sudden mortality in birds.

148. (D) Placing tissues in 10% neutral buffered formalin at a 10:1 ratio.
For legal or forensic histopathology in wildlife investigations, tissues must be preserved in ten percent neutral buffered formalin. A tissue to fixative volume ratio of at least one to ten is required to ensure complete penetration and rapid fixation of cells. This prevents autolysis, stabilizes cellular structures, and preserves tissues for subsequent histopathological evaluation.

149. (B) Mycobacterium tuberculosis
The presence of multifocal caseous nodules in the organs of a captive macaque is highly indicative of tuberculosis. The causative acid fast bacilli, Mycobacterium tuberculosis, infect alveolar macrophages, leading to chronic granulomatous inflammation. Confirming acid fast bacilli using the Ziehl-Neelsen staining method provides diagnostic proof of this zoonotic disease, requiring strict biosafety measures during necropsy.

150. (A) I, II, III and V
Nipah virus spills over from bats, SARS coronaviruses adapt in intermediate wildlife hosts, and Kyasanur Forest disease is transmitted by ticks. Avian influenza H5N1 can also spill over from wild birds to domestic poultry and humans. However, rabies virus is capable of infecting all warm blooded mammals, including domestic herbivores, meaning statement four is incorrect.

151. (D) Tyzzer's features intracellular silver-staining bacilli in parallel bundles within hepatocytes near necrotic margins.
Tyzzer's disease can be distinguished histopathologically from Salmonella by demonstrating intracellular bundles of Clostridium piliforme within hepatocytes near the margins of necrotic lesions. These rod-shaped, Gram-negative bacilli are highly argyrophilic and are best visualized using silver stains like Warthin-Starry. Conversely, Salmonella infection is characterized by focal histiocytic nodules rather than intracellular bacterial bundles.

152. (A) They lack L-gulonolactone oxidase, making them unable to synthesize ascorbic acid from glucose.
Guinea pigs are highly susceptible to scurvy because they completely lack the L-gulonolactone oxidase enzyme, which is required for the terminal step of ascorbic acid synthesis from glucose. Without this enzyme, they have an absolute dietary requirement for Vitamin C. Deficiency prevents normal collagen synthesis, leading to periarticular hemorrhages, swollen joints, and loose teeth.

153. (B) Anthrax
The postmortem combination of severe pulmonary edema, an enlarged, soft, friable spleen, and dark, unclotted blood exuding from orifices is diagnostic of anthrax. Detecting large, square ended, Gram-positive, capsulated bacilli in blood smears confirms Bacillus anthracis infection. Because opening the carcass triggers spore formation, suspected cases must not undergo a standard necropsy.

154. (B) Interstitial fibrosis, tubular dilation with protein casts, and glomerular sclerosis.
Chronic progressive nephropathy is a common age-related degenerative disease in laboratory rats. Histological hallmarks include marked interstitial fibrosis, tubular dilation filled with eosinophilic proteinaceous casts, tubular basement membrane thickening, and glomerular sclerosis. This progressive renal pathology is accelerated by high-protein diets, ultimately leading to chronic renal failure and uremia in older rodents.

155. (A) Rabbit hemorrhagic disease virus - Hepatic coagulation necrosis
Rabbit hemorrhagic disease virus is a highly contagious calicivirus that specifically targets hepatocytes. The primary and most characteristic histopathological lesion of this infection is acute, massive hepatic coagulation necrosis, often accompanied by disseminated intravascular coagulation. This rapid liver damage leads to

Click Here to Buy book on Amazon

138. (B) Most immunocompetent mice remain asymptomatic, but the virus can confound immunological research.
Exposure to murine norovirus in specific pathogen-free mouse colonies typically results in subclinical, asymptomatic infections in immunocompetent strains. However, because the virus replicates within dendritic cells and macrophages, it alters the host's immune responses and inflammatory pathways. This subclinical viral replication can significantly confound immunological research, leading to invalid or inconsistent experimental data.

139. (A) Acute poisoning causes rapid death with proventricular distension, while chronic causes emaciation and gall bladder distension.
Acute lead poisoning in waterfowl typically leads to rapid mortality, characterized postmortem by marked distension of the proventriculus with ingested food. In contrast, chronic lead toxicity allows animals to survive longer, presenting with profound emaciation, severe pectoral muscle atrophy, loss of subcutaneous fat, and conspicuous distension of the gall bladder with dark green bile.

140. (A) P-2, Q-1, R-4, S-3
Nipah virus is classified within the genus Henipavirus of the family Paramyxoviridae. Kyasanur Forest disease virus belongs to the family Flaviviridae. Canine distemper virus is categorized under the genus Morbillivirus in the Paramyxoviridae family. Elephant endotheliotropic herpesvirus is a member of the family Herpesviridae. These classifications reflect their structural and genomic characteristics.

141. (D) Chlamydia psittaci is an obligate intracellular bacterium that poses a significant zoonotic risk to humans.
Chlamydiosis, commonly known as psittacosis, is caused by Chlamydia psittaci, which is an obligate intracellular bacterium. The pathogen targets respiratory and systemic epithelial cells in wild and zoo birds, causing severe systemic disease. It is highly zoonotic, posing a significant infection risk to humans through the inhalation of dried feces or respiratory secretions.

142. (C) Viral destruction of rapidly dividing cells in the crypts of Lieberkühn, causing villous collapse.
Feline panleukopenia virus is a parvovirus that requires actively dividing cells in S phase for replication. It targets the mitotic crypt cells of Lieberkühn in the small intestine, leading to complete destruction of the mucosal epithelium. This cell loss prevents new enterocyte replacement, resulting in rapid villous collapse, severe hemorrhagic enteritis, and malabsorption.

143. (B) Pasteurella multocida
In laboratory rabbits, Pasteurella multocida is the primary etiology of otitis media and interna. The bacteria colonize the tympanic bulla, resulting in the accumulation of thick, caseous purulent exudate. This localized middle ear infection damages the vestibular apparatus, leading to the clinical presentation of torticollis, commonly referred to as head tilt or wry neck.

144. (D) Diffuse thinning of the long bones and multiple fractures
Hypovitaminosis A in captive birds primarily leads to squamous metaplasia of mucus secreting epithelial linings. This metaplastic change causes the formation of white pustules in the oral cavity, esophagus, and salivary glands, along with hyperkeratosis of the skin and feet. Diffuse thinning of long bones and multiple fractures are typical of metabolic bone disease, not hypovitaminosis A.

145. (A) The presence of distinct palatine tonsils
In the normal gross anatomy of a laboratory rat, palatine tonsils are entirely absent. Rats utilize other components of mucosa associated lymphoid tissue to perform local immune functions in the upper respiratory tract. Meanwhile, normal anatomical features of rats include the absence of a gall bladder, a large lobulated liver, and a prominent thymus in young adults.

NODIA

146. (C) Minimizing pursuit time, providing immediate physical cooling, and administering supportive fluid therapy.
To effectively prevent capture myopathy in wild herbivores, management interventions must focus on minimizing stress and physical exertion. Reducing pursuit time prevents severe metabolic acidosis and hyperthermia. Providing immediate physical cooling and administering supportive fluid therapy helps correct hyperthermia

164. (D) Non-suppurative encephalomyelitis with eosinophilic Negri bodies in the hippocampus or Purkinje cells.
The definitive histopathological finding of rabies in wild carnivores is nonsuppurative encephalomyelitis characterized by perivascular cuffing, microgliosis, and the presence of pathognomonic Negri bodies. These round or oval, eosinophilic intracytoplasmic inclusion bodies are primarily located within the pyramidal neurons of the hippocampus or the Purkinje cells of the cerebellum, representing sites of viral replication.

165. (B) Feline Panleukopenia
Feline panleukopenia virus infection in wild felids classically presents with severe, segmental to diffuse hemorrhagic enteritis, which histologically reveals necrosis of the crypts of Lieberkühn. Grossly, this damage manifests as prominent, dark red longitudinal mucosal bands, termed tiger-stripe hemorrhages, in the colon. These intestinal lesions are accompanied by marked lymphoid depletion in lymphoid organs.

166. (C) Newcastle is caused by a Paramyxovirus, while avian influenza is caused by an Orthomyxovirus.
Newcastle disease is caused by virulent strains of avian paramyxovirus serotype one, which belongs to the family Paramyxoviridae. In contrast, avian influenza is caused by influenza A viruses, which are members of the family Orthomyxoviridae. These two diseases are etiologically distinct RNA viruses, although they can present with overlapping clinical and pathological signs.

167. (D) It accelerates progression and severity of chronic progressive nephropathy.
In laboratory rats, diet formulation is a critical management factor affecting age-related diseases. High-protein and high-calorie diets accelerate the progression and increase the severity of chronic progressive nephropathy. Restricting protein and calorie intake in aging rats reduces renal workload and is highly effective in delaying the onset and limiting the pathological severity of kidney lesions.

168. (A) Severe hepatic lipidosis and an empty stomach in a late-gestation female
Pregnancy toxemia in guinea pigs is a metabolic disorder occurring during late gestation, primarily triggered by energy mismatch. The key postmortem findings of this disease are severe, diffuse hepatic lipidosis and an empty stomach in a late-gestation female. This represents a negative energy balance where rapid mobilization of body fat stores overwhelms the liver's metabolic capacity.

169. (B) Arsenic-based herbicide/pesticide
Arsenic poisoning in wild herbivores classically presents with bright green stomach contents, reflecting agricultural pesticide exposure, alongside severe hemorrhagic gastroenteritis and acute kidney injury. Arsenic compounds are highly cytotoxic, damaging mucosal capillaries and renal tubules. These dramatic necropsy findings differentiate arsenic toxicosis from other chemicals like anticoagulant rodenticides or organophosphates.

170. (A) Both A and R are true and R is the correct explanation of A.
Diclofenac administration in livestock led to a catastrophic decline in vulture populations. Vultures eating carcasses treated with diclofenac suffer severe toxicity because they lack the specific metabolic capacity to detoxify the drug. This deficiency causes uric acid accumulation, culminating in acute renal failure and fatal visceral gout, making both statements true and directly related.

171. (A) Sendai virus - Respiratory epithelium, causing pneumonia
Sendai virus is a highly infectious paramyxovirus in laboratory mice that primarily targets the respiratory epithelium, causing severe necrotizing bronchiolitis and interstitial pneumonia. In contrast, mousepox is an orthopoxvirus targeting the skin and spleen, murine hepatitis virus is a coronavirus targeting gastrointestinal or hepatic cells, and sialodacryoadenitis virus is a rat coronavirus targeting salivary and lacrimal glands.

172. (C) Destructive viral replication in capillary endothelial cells, causing hemorrhage and edema.
Elephant endotheliotropic herpesvirus causes high mortality in juvenile Asian elephants because of its specific tropism for vascular endothelial cells. Rapid, destructive replication of this virus within capillary endothelium damages vessel walls, leading to systemic vascular leakage, diffuse hemorrhage, pericardial effusion, and severe edema of the head, trunk, and limbs, culminating in acute hypovolemic shock.

Downloaded from www.pdf.tube

multi-organ hemorrhage and sudden death in susceptible European rabbits.

156. (C) Mycoplasmosis is caused by wall-less bacteria, whereas corynebacteriosis is caused by Gram-positive bacilli.
Murine mycoplasmosis is caused by Mycoplasma pulmonis, which are unique bacteria that completely lack a cell wall, making them resistant to beta-lactam antibiotics. In contrast, corynebacteriosis is caused by Corynebacterium kutscheri, which is a Gram-positive, rod-shaped bacterium. These cellular differences determine their respective pathogenic mechanisms and diagnostic criteria in laboratory rodent colonies.

157. (D) They inhibit acetylcholinesterase, leading to respiratory failure.
Organophosphate compounds irreversibly bind and inhibit acetylcholinesterase at synaptic junctions and neuromuscular endings. This inhibition leads to the accumulation of acetylcholine, causing continuous overstimulation of nicotinic and muscarinic receptors. In wild animals, this results in severe bronchoconstriction, excessive secretions, muscle tremors, and ultimately fatal respiratory failure from diaphragmatic muscle paralysis.

158. (C) Severe acute vesicular stomatitis
Tuberculosis in elephants is a chronic, progressive bacterial infection caused by Mycobacterium tuberculosis. Classic clinical and postmortem findings include chronic weight loss, poor body condition, and caseocalcareous lung granulomas. Detecting the bacteria via trunk wash cultures provides confirmation. Severe acute vesicular stomatitis is not associated with tuberculosis, typically indicating viral infections instead.

159. (B) Ophionyssus natricis (snake mite)
Inclusion body disease in captive boid snakes is strongly associated with infestations of the common snake mite, Ophionyssus natricis. This ectoparasite acts as a highly efficient mechanical vector for the causative reptarenaviruses, facilitating rapid transmission between cohoused or adjacent snakes. Controlling snake mite infestations represents a critical biosecurity measure in preventing disease spread.

160. (D) Don complete PPE → Use biosafety cabinet → Moisten carcass → Decontaminate surfaces and instruments
When performing a necropsy on a non-human primate, maximizing biosafety is essential because of potential zoonotic pathogens like Macacine herpesvirus one. Pathologists must don complete personal protective equipment and use a biosafety cabinet. Moistening the carcass prevents aerosolization of infectious particles, and subsequent thorough decontamination of surfaces and instruments minimizes occupational transmission risks.

NODIA

161. (A) The animal was infected with the dimorphic fungus Coccidioides immitis.
Granulomatous pneumonia characterized by the presence of large, thick-walled spherules containing numerous small endospores is the classic histopathological hallmark of coccidioidomycosis. This systemic disease is caused by the soil-dwelling dimorphic fungus Coccidioides immitis. Animals contract the infection by inhaling airborne arthroconidia from dry soils, leading to chronic pulmonary or disseminated disease.

162. (C) The disease typically causes a high mortality rate in adult populations.
Foot-and-mouth disease, caused by an Aphthovirus, affects wild ruminants, presenting with vesicular lesions on the mouth and feet. While morbidity is high, mortality in adult animals is typically low. However, young ruminants can suffer high mortality due to acute myocarditis, historically referred to as tiger heart, because of the striped appearance of necrotic cardiac muscle.

163. (A) Visceral gout involves urate deposition on the serosal surfaces of internal organs (pericardium, liver), while articular gout involves urate tophi in the joints.
In birds, visceral gout is characterized by the rapid precipitation of chalky white monosodium urate crystals onto the serosal surfaces of visceral organs, particularly the pericardium and liver. Articular gout, conversely, is a more chronic condition where urate tophi deposit within joint spaces and synovial sheaths, leading to severe localized inflammation and periarticular swelling.

129. (A) Syncytial cells with eosinophilic intracytoplasmic and intranuclear inclusions in respiratory and intestinal epithelia.
Peste des petits ruminants, caused by a small ruminant morbillivirus, is characterized histopathologically by the presence of multinucleated syncytial cells containing both eosinophilic intracytoplasmic and intranuclear inclusion bodies. These diagnostic inclusions are primarily found within the damaged epithelial tissues of the respiratory tract, digestive tract, and lymphoid organs of affected wild and domestic small ruminants.

130. (D) A is false but R is true.
Performing a necropsy on a captive reptile carries a significant risk of zoonotic disease transmission, particularly from Salmonella species. Reptiles are well-documented, highly prevalent asymptomatic carriers of Salmonella within their gastrointestinal tract. Consequently, the assertion that zoonotic transmission is a negligible risk is false, while the reasoning regarding their role as reservoir hosts is true.

131. (A) Murine Mycoplasmosis - Serology and PCR
In laboratory rodent colonies, routine health surveillance for murine mycoplasmosis, caused by Mycoplasma pulmonis, relies on serology testing and polymerase chain reaction assays. This molecular and serological testing provides highly sensitive and specific detection. Other pathogens, like the pinworm Syphacia obvelata, are diagnosed using perianal tape tests, rather than standard enzyme linked immunosorbent assays.

132. (D) Respiratory failure from ascending paralysis
Tick paralysis is caused by salivary neurotoxins injected by certain feeding ticks, such as Dermacentor species. The toxin inhibits acetylcholine release at the neuromuscular junction, leading to a progressive, flaccid, ascending motor paralysis. If untreated, this ascending paralysis eventually reaches the diaphragm and intercostal muscles, causing fatal respiratory failure in affected wild carnivores.

133. (B) The animal likely has Macacine herpesvirus 1 and requires extreme biosafety.
A rhesus macaque presenting with oral vesicles and ulcers is highly suspected of harboring Macacine herpesvirus one, also known as B virus. While this alphaherpesvirus causes only mild, self limiting mucosal lesions in its natural macaque hosts, it is extremely pathogenic in humans, causing fatal encephalomyelitis. Consequently, any suspected case warrants the highest level of biosafety precautions.

134. (C) Osteomyelitis and systemic septicemia
Untreated infectious stomatitis, or mouth rot, in captive snakes often progresses from localized oral mucosal inflammation to deep tissue invasion. Opportunistic oral bacteria, such as Pseudomonas or Aeromonas species, can invade the mandibular or maxillary bones, leading to severe osteomyelitis. From there, the pathogens readily enter the bloodstream, causing fatal systemic septicemia.

135. (D) Pasteurella or Mannheimia species
In young wild bighorn sheep, severe, acute, fibrinohemorrhagic pleuropneumonia is typically caused by respiratory bacterial pathogens of the Pasteurellaceae family, specifically Pasteurella multocida or Mannheimia haemolytica. These bacteria, often transmitted from domestic sheep, cause rapid, devastating lung consolidation and fibrinous pleuritis, which represents a primary driver of wild bighorn herd die offs.

136. (A) A history of consuming calcium-dusted, UV-exposed diet
Nutritional secondary hyperparathyroidism in reptiles is caused by inadequate dietary calcium intake, excess dietary phosphorus, or a lack of ultraviolet B radiation needed for active vitamin D synthesis. Therefore, a documented history of consuming a properly calcium dusted diet along with adequate ultraviolet light exposure effectively eliminates this metabolic bone disease as a primary diagnosis.

137. (B) KFD is a tick-borne flavivirus causing hemorrhagic fever in humans and mortality in monkeys.
Kyasanur Forest disease is caused by a tick-borne virus belonging to the Flaviviridae family. It is transmitted primarily by the bite of infected Haemaphysalis ticks. The disease causes severe, acute hemorrhagic fever in humans and is highly fatal in non-human primates, particularly langurs and bonnet macaques, serving as a sentinel indicator in endemic forest regions.

Click Here to Buy book on Flipkart

173. (D) The reptile has nutritional secondary hyperparathyroidism due to poor diet or low UV exposure.
A captive reptile presenting with soft, pliable mandibles, commonly known as rubber jaw, and multiple spontaneous fractures of the long bones is exhibiting classic signs of nutritional secondary hyperparathyroidism. This metabolic bone disease is caused by an inadequate dietary calcium to phosphorus ratio or insufficient ultraviolet light exposure, which prevents vitamin D synthesis.

174. (C) Secondary bacterial infection causing otitis media, torticollis, and meningoencephalitis.
Untreated infestation of the ear mite, Psoroptes cuniculi, in rabbits can cause severe, painful hyperkeratotic crusts in the external ear canal. If the tympanic membrane is breached, the inflammatory process and secondary bacterial pathogens can invade the middle and inner ear. This progression leads to otitis media, torticollis, and potentially fatal meningoencephalitis.

175. (B) Hepatic coccidiosis
Hepatic coccidiosis in rabbits is caused by the apicomplexan protozoan Eimeria stiedae. The parasite infects and replicates within the biliary epithelial cells, leading to marked biliary hyperplasia, thickened bile ducts, and the presence of numerous developmental protozoal stages on histopathology. This liver pathology is distinct from bacterial infections like Tyzzer's disease or viral diseases.

176. (D) The presence of massive hepatic necrosis and coagulation with no respiratory lesions.
In rabbits, the presence of massive hepatic coagulation necrosis and disseminated intravascular coagulation with no respiratory system involvement is indicative of rabbit hemorrhagic disease virus, effectively eliminating pasteurellosis. Pasteurella multocida typically causes severe respiratory lesions, including mucopurulent rhinitis, fibrinous pleuropneumonia, middle ear infections, or subcutaneous abscesses, rather than isolated, massive hepatic necrosis.

177. (A) It is a tick-borne protozoal disease destroying erythrocytes, leading to anemia and icterus.
Babesiosis is a tick-borne disease caused by protozoan parasites of the genus Babesia, which infect and replicate within erythrocytes. This intraerythrocytic replication leads to intravascular and extravascular hemolysis. In wild canids, the resulting red blood cell destruction causes severe clinical signs such as regenerative hemolytic anemia, hemoglobinuria, splenomegaly, and icterus.

178. (C) Cervical swelling, porphyrin staining ("red tears"), and photophobia, resolving in a few weeks but disrupting research.
Sialodacryoadenitis virus is a highly contagious coronavirus of laboratory rats that targets the salivary and lacrimal glands. If a barrier system fails, exposed rats develop transient cervical swelling, porphyrin-stained secretions around the eyes referred to as red tears, and photophobia. Although the infection is self-limiting and resolves within weeks, it significantly confounds research.

179. (A) Mousepox causes cutaneous lesions, extremity necrosis, and splenic necrosis, while MHV targets GI or liver, forming syncytia.
Mousepox, caused by ectromelia virus, is an orthopoxvirus that causes cutaneous rash, necrosis of extremities, and focal necrosis in the spleen and liver. Conversely, mouse hepatitis virus is a coronavirus targeting the enteric mucosa or hepatic parenchyma, characterized histopathologically by multinucleated syncytia. These distinct lesion profiles easily differentiate the two viral diseases at necropsy.

180. (A) P-2, Q-4, R-1, S-3
In guinea pigs, scurvy causes periarticular hemorrhages and enlarged costochondral junctions. Johne's disease in wild ruminants causes a corrugated, thickened intestinal mucosa. Tyzzer's disease in mice leads to miliary hepatic necrosis. Myxomatosis in rabbits is characterized by gelatinous subcutaneous skin nodules. These classical gross lesions provide a reliable basis for tentative postmortem diagnoses.

181. (B) Chronic infection leads to right-sided heart failure, hypertension, and vena cava syndrome.
Chronic Dirofilaria immitis infection in wild canids leads to significant mechanical obstruction within the pulmonary arteries and right ventricle. This obstruction causes pulmonary hypertension and right-sided congestive heart failure. In severe cases, heavy worm burdens can retrograde into the vena cava, producing vena cava syndrome

Downloaded from www.pdf.tube

200. (A) V
Capture myopathy is caused by extreme physical exertion, fear, and muscle overactivity during handling or translocation of wild animals, leading to metabolic acidosis and muscle necrosis. It is not caused by Vitamin D deficiency. Meanwhile, anthrax, canine distemper, elephant endotheliotropic herpesvirus, and lead poisoning are correctly associated with their respective classical pathological features.

201. (B) The animal is infected with Mycobacterium tuberculosis, posing a severe zoonotic risk.
A captive macaque presenting with progressive weight loss, coughing, and multiple caseous lung granulomas is highly likely to be infected with Mycobacterium tuberculosis. Non-human primates are extremely susceptible to tuberculosis, which is a major zoonotic threat. Suspected cases require strict isolation, specialized biosafety protocols during necropsy, and immediate reporting to protect staff.

202. (B) The primary lesion is widespread cortical necrosis and severe malacia.
Rabies virus is a highly neurotropic pathogen that does not cause widespread cortical necrosis or gross cerebral malacia. Instead, the infection is characterized by nonsuppurative encephalomyelitis, perivascular cuffing, microgliosis, and pathognomonic eosinophilic intracytoplasmic Negri bodies. The virus is transmitted via saliva and moves retrograde along peripheral nerves to the central nervous system.

203. (A) SPF animals lack specified pathogens with undefined flora, while gnotobiotic animals have a completely defined flora.
Specific pathogen-free laboratory animals are guaranteed to be free of particular listed pathogens, but they possess a normal, undefined microflora. In contrast, gnotobiotic animals are maintained in sterile isolators and possess a completely defined, known microflora, or are completely germ-free. This basic distinction determines their suitability for different types of microbiological research.

204. (C) Multiple, miliary, pale necrotic foci scattered throughout the hepatic parenchyma
At necropsy, the classical gross pathological finding of Tyzzer's disease, caused by Clostridium piliforme, in laboratory rodents is the presence of multiple, miliary, pale necrotic foci scattered throughout the hepatic parenchyma. This liver pathology is typically accompanied by necrotizing ileocolitis and myocardial necrosis, forming a characteristic triad of lesions for this bacterial infection.

205. (D) Feline Panleukopenia
A captive tiger presenting with severe, acute hemorrhagic enteritis and complete destruction of the intestinal crypts is suffering from feline panleukopenia. The causative parvovirus requires mitotic cells in S phase for replication, leading to direct destruction of rapidly dividing crypt enterocytes of Lieberkühn. This causes mucosal collapse, severe clinical diarrhea, dehydration, and high mortality.

NODIA

206. (B) Large, eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies) in epithelial cells
The cutaneous, or dry, form of avian pox in wild birds is characterized histopathologically by severe epithelial hyperplasia and the presence of Bollinger bodies. These are large, eosinophilic intracytoplasmic inclusion bodies within infected epidermal cells. Finding these inclusions in hyperplastic skin lesions provides a definitive diagnosis of this avipoxvirus infection during postmortem evaluation.

207. (B) Stimulating parathyroid hormone release, causing bone resorption and secondary hyperparathyroidism.
In captive reptiles, an inverted calcium-to-phosphorus ratio stimulates the parathyroid glands to release parathyroid hormone. This excess hormone triggers systemic bone resorption to normalize blood calcium levels, culminating in nutritional secondary hyperparathyroidism. This metabolic bone disease is characterized by pliable mandibles, swollen long bones, and spontaneous fractures, rather than soft tissue mineralization.

208. (D) Fibrinous airsacculitis, hepatosplenomegaly, and elementary bodies in macrophages.
Avian chlamydiosis, caused by Chlamydia psittaci, presents grossly with fibrinous airsacculitis, fibrinous pericarditis, hepatomegaly, and splenomegaly. Histopathology and cytology reveal characteristic basophilic or eosinophilic elementary bodies clustered within the cytoplasm of infected macrophages. These findings provide a definitive basis for diagnosing this highly

STUDYING FROM PDFs?

THINK AGAIN.

If you are studying only from PDFs,
you are wasting valuable time.

Research shows that learning from

PRINTED BOOKS has up to

4× HIGHER RETENTION

than studying from screens.

PRINT BOOK = BETTER MEMORY

PDF = LOWER RETENTION

Those who study from **PRINTED BOOKS**
move **AHEAD.**

Others are left **BEHIND**
in competition.

In competitive exams,
being average is not enough.

STAY ONE STEP AHEAD.

191. (B) Stomatitis causes oral petechiae, caseous exudate, and ulcers, while inclusion body disease causes neurological signs and inclusions.
In captive snakes, infectious stomatitis presents with localized oral mucosal petechiae, thick caseous exudates, and ulcerative lesions. In contrast, inclusion body disease is a systemic arenavirus infection. It is characterized clinically by progressive neurological signs like stargazing or loss of righting reflex, and histopathologically by eosinophilic intracytoplasmic inclusions in the brain and other tissues.

192. (D) Muscle necrosis releases myoglobin into the blood, which precipitates in and damages renal tubules.
Exertional rhabdomyolysis in translocated wild herbivores causes severe, acute necrosis of skeletal muscle tissue. This widespread myofiber breakdown releases massive quantities of myoglobin into the systemic circulation. In the kidneys, this high concentration of myoglobin precipitates within the renal tubules, leading to direct tubular epithelial cytotoxicity, cast obstruction, and acute renal failure.

193. (A) Diclofenac
Diclofenac is a non-steroidal anti-inflammatory drug widely used in livestock that is highly toxic to Gyps vultures. Vultures feeding on treated carcasses absorb the drug, which inhibits renal blood flow and causes acute renal tubular necrosis. This prevents normal uric acid excretion, culminating in hyperuricemia and fatal visceral gout across multiple organs.

194. (C) Depletion of lymphoid tissue and necrosis of intestinal crypt epithelium.
Histopathological examination of a wild felid with feline panleukopenia reveals marked necrosis of the intestinal crypt epithelium within the small intestine, leading to villous atrophy. Additionally, because the parvovirus targets rapidly dividing cells, there is severe lymphoid depletion in the spleen, lymph nodes, and thymus, alongside myeloid hypoplasia in the bone marrow.

195. (A) Vitamin C deficiency - Defective collagen synthesis and periarticular hemorrhages.
Vitamin C deficiency in guinea pigs blocks the hydroxylation of proline and lysine, leading to defective collagen synthesis. This structural weakness in blood vessels and connective tissues results in periarticular hemorrhages, joint swelling, and skeletal deformities. This pathogenesis is unique to guinea pigs and primates due to their shared lack of L-gulonolactone oxidase.

196. (D) RHD is a calicivirus causing hepatic necrosis, while Myxomatosis is a poxvirus causing skin tumors.
Rabbit hemorrhagic disease is caused by a calicivirus that specifically targets hepatocytes, resulting in acute, massive hepatic coagulation necrosis and severe hemorrhages. In contrast, myxomatosis is caused by a poxvirus that induces benign, gelatinous mucinous skin tumors and subcutaneous swelling around the face and eyes. These two major rabbit pathogens belong to entirely different viral families.

197. (B) It disrupts enzyme systems involved in heme synthesis, causing anemia, neurotoxicity, and nephrotoxicity.
Lead poisoning disrupts cell metabolism by binding to sulfhydryl groups on essential enzymes, particularly delta-aminolevulinic acid dehydratase and ferrochelatase, which are required for heme synthesis. This inhibition leads to regenerative anemia, severe neurotoxicity due to altered neurotransmitter release, and nephrotoxicity characterized by pathognomonic acid-fast intranuclear inclusion bodies within the renal tubules.

198. (C) Vesicular lesions on the coronary bands
Peste des petits ruminants in wild goats is characterized by erosive and ulcerative stomatitis, gastroenteritis, progressive bronchointerstitial pneumonia, and multinucleated syncytial cells. Vesicular lesions on the coronary bands are not associated with this morbillivirus infection, instead being classic indicators of vesicular diseases such as foot-and-mouth disease or vesicular stomatitis in wild populations.

199. (D) Feeding diets high in protein and calories
In laboratory rats, chronic progressive nephropathy is a major spontaneous degenerative disease. Its onset and severity are highly influenced by dietary factors. Feeding rats diets containing excessive protein and energy levels accelerates glomerular damage and tubular degeneration, worsening renal pathology. Managing protein and caloric intake remains the most effective way to slow disease progression.

Click Here to Buy book on Amazon

PAPERGPT
125, Sector 6, Vidhyadhar Nagar, Jaipur 302039

MRP. ₹ 660

ASRB EXAM 2026

VETERINARY PATHOLOGY

NODIA

Scan QR For Demo PDF

zoonotic, obligate intracellular bacterial infection in birds.

209. (A) Anticoagulant rodenticide
Anticoagulant rodenticide poisoning in wild canids classically causes severe hemorrhaging into body cavities due to the inhibition of vitamin K epoxide reductase. This block prevents active coagulation factors from forming, leading to fatal blood loss. Postmortem findings like massive hemothorax or hemoperitoneum are highly characteristic of exposure to these common chemical pest control agents.

210. (C) A is true but R is false.
Specific pathogen-free rodents exposed to normal environmental flora can suffer severe clinical disease and mortality because they are immunologically naive to common environmental agents. However, they are not immunodeficient, possessing completely functional T and B lymphocytes. Therefore, the assertion that they may suffer severe disease is true, while the reasoning regarding genetic immunodeficiency is false.

****

Downloaded from www.pdf.tube

characterized by acute intravascular hemolysis and hepatic dysfunction.

182. (C) Hematogenous bacterial spread from the GI tract, causing embolic pneumonia and abscesses.
In laboratory rodents, Corynebacterium kutscheri is the causative agent of pseudotuberculosis. The bacteria usually reside subclinically in the gastrointestinal tract or lymph nodes. During periods of stress or immunosuppression, the pathogens enter the bloodstream, resulting in hematogenous dissemination. This embolic spread leads to the formation of multiple caseous abscesses and bronchopneumonia in the lungs.

183. (D) Mycobacterium paratuberculosis
Johne's disease in wild ruminants and some wild felines is caused by Mycobacterium avium subspecies paratuberculosis. The chronic infection manifests postmortem as a markedly thickened, corrugated intestinal mucosa and enlarged, pale mesenteric lymph nodes. Histopathology reveals abundant acid-fast bacilli residing within the cytoplasm of epithelioid macrophages located in the lamina propria.

184. (A) Massive hepatic necrosis and hemorrhage
Lead poisoning in free-ranging birds classically causes proventricular and gizzard impaction, severe pectoral muscle atrophy, and acid-fast intranuclear inclusion bodies within renal tubular epithelial cells. These inclusions represent lead-protein complexes. While the liver may show some degenerative changes or congestion, massive hepatic necrosis and hemorrhage are not characteristic postmortem findings of lead toxicosis.

185. (B) The lack of a gall bladder
The normal gross anatomy of a laboratory mouse features a well-developed gall bladder, making its absence an anatomical exception. This is in contrast to laboratory rats, which normally lack a gall bladder. Other standard anatomical features of mice include a prominent pale forestomach, extensive interscapular brown fat, and large lobulated seminal vesicles in males.

186. (B) HPLC or mass spectrometry for specific anticoagulant compounds
To definitively confirm anticoagulant rodenticide toxicity in a deceased wild carnivore, high-performance liquid chromatography or mass spectrometry must be performed on liver tissue samples. These highly sensitive analytical techniques directly detect and quantify specific rodenticide compounds. Since the liver is the primary site of accumulation for these toxicants, it is the ideal diagnostic specimen.

187. (A) Waterfowl are primary reservoirs and usually show severe signs of highly pathogenic strains.
While waterfowl are the primary natural reservoirs for low pathogenic avian influenza viruses, they typically remain completely asymptomatic when infected. Most waterfowl strains do not cause severe disease in these wild hosts, even when highly pathogenic. This allows the birds to migrate and shed the virus extensively, spreading it to domestic poultry populations.

188. (C) Mixing feces with 10% polyvinyl alcohol or refrigerating fresh samples.
For parasitological examination of wildlife fecal samples, preservation techniques must keep parasitic ova, cysts, and trophozoites intact. Mixing the feces with ten percent polyvinyl alcohol stabilizes delicate protozoal structures. Alternatively, refrigerating fresh fecal specimens preserves helminthes and protozoan stages, preventing egg hatching and developmental progression that would confound diagnostic identification.

189. (D) Visceral gout
A captive raptor presenting with multiple chalky white nodules on the epicardium, liver, and air sacs has visceral gout. This condition is caused by hyperuricemia resulting from dehydration and an excessive dietary protein intake. The kidneys fail to excrete the high nitrogenous load, leading to the rapid precipitation of monosodium urate crystals on internal organs.

NODIA

190. (A) I, II and III
Nipah virus causes severe encephalitis in humans. Macacine herpesvirus one is highly zoonotic and fatal in humans but causes mild oral lesions in rhesus macaques. Rabies is a fatal viral encephalitis in humans once clinical signs manifest. However, avian influenza H5N1 can infect humans, and non-human primate tuberculosis poses a significant risk to people.